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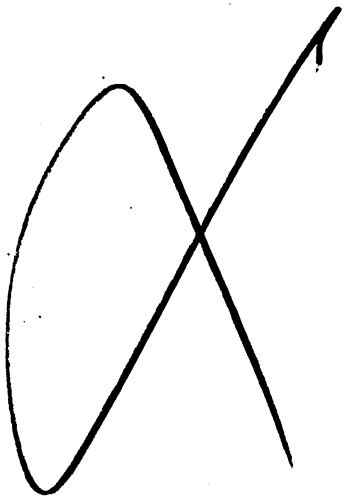

TRANSACTIONS

OF THE

EPIDEMIOLOGICAL SOCIETY

OF

LONDON.

NEW SERIES. VOL. VI.

SESSION 1886-87.

LONDON:
SHAW AND SONS, FETTER LANE, E.C.

MDCCCLXXXVIII.

LONDON:
WHITING AND COMPANY, SARDINIA STREET, LINCOLN'S INN FIELDS.

CONTENTS.

EPIDEMIOLOGICAL SOCIETY OF LONDON.

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FOR THE SESSION 1886-7.

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Vice-Presidents.

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Sir HENRY W. ACLAND, K.C.B., M.D., F.R.S., Regius Professor of Medicine in the University of Oxford.
Sir WILLIAM JENNER, Bart., K.C.B., M.D., D.C.L., F.R.S., Physician in Ordinary to Her Majesty the Queen.
BENJAMIN W. RICHARDSON, M.D., F.R.S.
ROBERT LAWSON, Inspector-General of Hospitals, Royal Army (*retired*).
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GEORGE BUCHANAN, M.D., F.R.S.
J. BURDON SANDERSON, M.D., F.R.S.

Treasurer.

R. THORNE THORNE, M.B., F.R.C.P., 45, Inverness Terrace, W.

General Secretaries.

SHIRLEY F. MURPHY, 41, Queen Anne Street, W.
CHARLES E. PAGET, Kendal, Westmorland.

Secretaries for Navy.

WALTER DICKSON, M.D., R.N., Medical Officer to the Hon. Board of Customs, Custom House, E.C.
W. H. LLOYD, M.D., Dept. Inspector-Gen. R.N.

Secretary for Army.

ROBERT LAWSON, Inspector-General of Hospitals (*retired*).

Foreign and Colonial Secretaries.

Germany and Russia	ALEXANDER COLLIE, M.D.
Sweden, Norway, and Denmark ...	J. W. MOORE, M.D.
Portugal and the Brazils ...	J. J. L. DONNET, M.D., R.N.
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West Indies and South America ...	G. C. HENDERSON, M.D.
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Indian Ocean and East Africa ...	J. CHRISTIE, M.D.
North America	JOSEPH EWART, M.D.
Polynesia	BOLTON G. CORNEY.

Other Members of Council.

J. WORTLEY AXE, F.R.C.V.S.
ROBERT CORY, M.D.
W. H. CORFIELD, M.D.
Surg.-Gen. C. A. GORDON, C.B., M.D., Q.H.P.
EDWIN HAWARD, M.D.
G. B. LONGSTAFF, M.B.
Sir A. D. HOME, K.C.B., V.C., M.D.
Sir W. GUYER HUNTER, K.C.M.G., M.D., M.P.
JOHN MACCOMBIE, M.D.
Surg.-Major R. PRINGLE, M.D.
HERMANN WEBER, M.D.

LIST OF MEMBERS.*

(P) *President;* (T) *Treasurer;* (S) *Secretary.*

HONORARY MEMBERS.

Elected.
1867. Acland, Sir Henry W., K.C.B., M.D., F.R.S., Oxford.
1868. Guipon, D. J., Laon Aisne, France.
1885. Humphreys, Noel, General Register Office, Somerset House.
1852. Richardson, B. W., M.D., F.R.S., 25, Manchester Square, W.
1850. Simon, Sir John, K.C.B., D.C.L., F.R.S., 40, Kensington Square, W.
1883. Sonsino, Prospero, M.D., 11, Via San Lorenzo, Pisa.
1863. Stark, Jas., M.D., Underwood, Bridge of Allan, Stirlingshire, (*retired*).

ORDINARY MEMBERS.

1887. Abraham, P. S., M.D., 11, Nottingham Place, W.
1878. Airy, Hubert, M.D., Local Government Board, Whitehall, S.W.
1882. Armstrong, Henry E., 6, Wentworth Place, Newcastle-on-Tyne.
1884. Atkinson, F. E., Whitefriars, Settle, Yorkshire.
1881. Axe, J. Wortley, F.R.C.V.S., Veterinary College, Camden Town, N.W.
1882. Barry, F. W., M.D., Local Government Board, Whitehall, S.W.
1870. Buchanan, George, M.D., F.R.S., 24, Nottingham Place, W. (P. 1881-83. T. 1871-81.)
1886. Cameron, James, M.D., Guildford House, Hendon, W.
1882. Carpenter, Alfred, M.D., Duppas House, Croydon.

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* Members are requested to inform the Secretaries of any corrections when necessary.

Elected.

1885. Cobb, R., Surg., 15, Matham Grove, East Dulwich, S.E.
1868. Cock, Fred., M.D., 1, Westbourne Park Terrace, W.
1875. Collie, Alexander, M.D., Eastern District Hospital, Homerton, E.
1887. Collins, G. W., 66, Adelaide Road, N.W.
1884. Comyn, J. I., Dep. Surg.-Gen., 32, Dawson Place, W.
1871. Corfield, W. H., M.D., 19, Savile Row, W. (S. 1871-72.)
1885. Cornish, W. R., Surg.-Gen., 8, Cresswell Gardens, South Kensington, S.W.
1879. Cory, Robert, M.D., 73, Lambeth Palace Road, S.E.
1863. Crawford, Sir Thomas, K.C.B., M.D., Director-General Army Med. Dept., Craig's Court, S.W.
1887. Cresswell, Francis, Winchmore Hill, N.
1884. Cullimore, D. H., M.D., 27, Welbeck Street, W.
1863. Dickson, Walter, M.D., R.N., Fairfield, Belvedere Road, Upper Norwood. (President 1885-87.)
1886. Donovan, Justin F., M.D., R.N., Malta.
1880. Duka, Theodore, M.D., 55, Nevern Square, S.W.
1879. Ewart, Joseph, M.D., Dep. Surg.-Gen. Ind. Med. Dept. (*retired*), Montpelier Hall, Brighton.
1884. Faulkner, Alex., Surg., Messrs. Grindley & Co., 55, Parliament Street, S.W.
1875. Fayrer, Sir Joseph, K.C.S.I., M.D., LL.D., F.R.S., Surg.-Gen. Ind. Med. Dept. (*retired*), 53, Wimpole Street, W. (P. 1879-81.)
1883. Furnivall, C. H., Lynton Road, Acton, Middlesex.
1880. Gordon, C. A., C.B., M.D., Q.H.P., Surg.-Gen., 25, Westbourne Square, W.
1885. Gresswell, D. Astley, B.A., M.B., Local Government Board, Whitehall, S.W.
1884. Hanbury, Sir James, K.C.B., Surg.-Gen. (*Gone abroad.*)
1875. Hare, C. J., M.D., Berkeley House, 15, Manchester Square, W.
1885. Harris, A. E., L.R.C.P., 14, The Avenue, Sunderland.
1884. Harris, F. W. H. D., Station Hospital, Madras.
1860. Haward, Edwin, M.D., 9, Harley Street, W.
1882. Hobley, S. Halford, 49, Bellevue Road, Southampton.
1883. Hopwood, E. O., M.D., London Fever Hospital, Islington, N.
1887. Hunt, J. Percival, Sheerness Hospital, Kent.
1880. Hunter, Sir W. Guyer, K.C.M.G., M.D., M.P., Surg.-Gen. Ind. Med. Dept., 21, Norfolk Crescent, W.
1887. James, C. A., 24, Cazenove Road, Stoke Newington, N.
1863. Jenner, Sir William, Bart., K.C.B., M.D., F.R.S., 63, Brook Street, W. (P. 1866-68.)
1881. Kennett-Barrington, Sir V. H. B., 15, Hyde Park Gardens, W.
1881. Kynsey, W. R., Colombo, Ceylon.
1863. Lawson, Robert, Inspector-Gen., 20, Lansdowne Road, Notting Hill, W. (P. 1871-73.)
1886. Lingard, Alfred, 49, Lambeth Palace Road, S.E.
1884. Ligertwood, Thomas, M.D., Dep. Surg.-Gen., Royal Hospital, Chelsea, S.W.
1885. Lloyd, W. Harris, M.D., Dep. Inspector-General, 4, Alfred Place West, South Kensington, S.W.
1880. Longstaff, G. B., M.B., Southfield Grange, West Hill Road, Wandsworth, S.W.
1882. Manifold, M. F., Surg.-Gen., 42, Cathcart Road, South Kensington, S.W.
1885. Martin, Sydney, M.D., 135, Gower Street, W.C.

Elected.

1878. MacCombie, John, M.D., South-Eastern District Hospital, Deptford, S.E.
1880. McKellar, A. Oberlin, St. Thomas's Hospital, S.E.
1885. McLeod, K., A.M., M.D., Surgeon-Major, Messrs. Grindley and Co., 55, Parliament Street, S.W.
1885. Moir, John, Hack Road, Victoria Docks, E.
1883. Moore, Sandford, M.B., Surg.-Major, 33, Clarendon Park West, Kingstown.
1872. Murray, John, M.D., Surg.-Gen. Ind. Med. Dept. (*retired*), 17, Westbourne Square, W. (P. 1877-79.)
1875. Murphy, Shirley Forster, Hon. Secretary, 41, Queen Anne Street, Cavendish Square, W. (S. from 1877.)
1883. Nash, W., M.B., Surg.-Major Army Med. Dept., Craig's Court, S.W.
1885. Page, David, M.D., Local Government Board, Whitehall, S.W.
1883. Paget, Charles E., Hon. Secretary, Kendal, Westmorland. (S. from 1883.)
1858. Paine, H. J., M.D., Elmsfield, Newport Road, Cardiff, South Wales.
1884. Parkes, Louis, M.D., 65, Cadogan Square, S.W.
1883. Parsons, H. Franklin, M.D., Local Government Board, Whitehall, S.W.
1880. Payne, J. F., M.D., 78, Wimpole Street, W.
1884. Pringle, R., Surg.-Major, M.D., 2, Aberdeen Terrace, Blackheath, S.E.
1885. Redmayne, Hugh, Ambleside, Westmorland.
1880. Reid, Sir John Watt, K.C.B., M.D., Director-General Navy Med. Dept., Admiralty, Northumberland Avenue, S.W.
1875. Reynolds, J. Russell, M.D., F.R.S., 38, Grosvenor Street, W.
1875. Roberts, F. T., M.D., 102, Harley Street, W.
1873. Saunders, C. E., M.D., 21, Lower Seymour Street, W.
1882. Scriven, J. B., Brigade-Surgeon Ind. Med. Dept., 95, Oxford Gardens, Notting Hill, W.
1873. Seaton, E. C., M.D., 35, George Street, Hanover Square, W.
1884. Simpson, W. J., M.D., Bengal Club, Calcutta.
1878. Smee, A. H., The Grange, Hackbridge, Surrey.
1885. Smith, D. B., Dep. Surg.-Gen., East Cliff, Woolston, near Southampton.
1882. Smith, W. R., M.D., F.R.S.E., 74, Great Russell Street, Bloomsbury, W.C.
1880. Spear, John, Local Government Board, Whitehall, S.W.
1872. Squire, William, M.D., 6, Orchard Street, W. (S. 1873-77.)
1880. Sweeting, Richard, Western District Hospital, Fulham, S.W. (T. 1887.)
1886. Taylor, M. W., M.D., Kenmure House, 202, Earl's Court Road, S.W.
1871. Thorne, R. Thorne, M.B., F.R.C.P., 45, Inverness Terrace, W. (T. from 1881-87. S. 1879-81. P. 1887.)
1876. Turner, George, M.D., High Street, Hoddesden, Herts.
1854. Weber, Hermann, M.D., 10, Grosvenor Street, W.
1882. Whitelegge, B. A., M.D., 13, Belgrave Square, Nottingham.
1883. Willoughby, Edward F., M.D., Bratton Lodge, Green Lanes, N.
1887. Yarrow, G. E., M.D., 87, Old Street, E.C.
1883. Young, Keith Downes, A.R.I.B.A., 17, Southampton Street, Bloomsbury, W.C.

CORRESPONDING AND NON-RESIDENT MEMBERS.

Elected.

N.R. 1867. Allbutt, T. Clifford, M.D., 6, Park Square, Leeds.
C. 1875. Arkhangelsky, D., M.D., St. Petersburg, Russia.
N.R. 1875. Armistead, William, M.B., Great Shelford, Cambridge.
C. 1852. Barker, R. H., Hungerford, Berks.
C. 1852. Bartolomé, Mariano Martin de, 334, Glossop Road, Sheffield.
C. 1856. Barton, E. H., New Orleans, U.S.A.
C. 1853. Beneke, F. W., Marburg, Germany.
N.R. 1883. Bovill, Edward, M.B., C.M., Motihari, Champaran, India.
C. 1860. Brionde, A. de, Haute Loire, France.
N.R. 1864. Buckle, Fleetwood, M.D.
N.R. 1866. Caddy, J. T., M.D., Dep. Inspec. Gen., 82, Redcliffe Square, S.W.
N.R. 1881. Carter, H. Vandyke, M.D., Surg.-Major Ind. Med. Dept.
C. 1851. Cartledge, Benjamin, Vet. Surg., Sheffield.
N.R. 1883. Castor, G. F., M.B., Colonial Surgeon, Cape Coast Castle, West Coast of Africa.
C. 1871. Christie, James, M.D., Zanzibar.
N.R. 1873. Close, C. Stratherne, Surgeon-Major Army Med. Dept.
N.R. 1875. Coffin, R. J. M., Alwington House, Baron's Court, West Kensington, S.W.
C. 1875. Colvill, W. H., Surg.-Major, Bagdad.
N.R. 1880. Condon, J. H., M.D., Surg.-Major Ind. Med. Dept., Cawnpore, N.W.P. India.
C. 1851. Cooke, R. T. E. B., The Haven, Esplanade Gardens, Scarborough.
N.R. 1885. Corney, Bolton G., Suva, Fiji.
C. 1856. Cox, Wm. Isidore, Hawkesbury, Upton, Chipping-Sodbury, Gloucestershire.
N.R. 1885. Crawford, D. G., Surg. I.M.S., Mymensingh, Lower Bengal.
C. 1871. Cuningham, J. M., M.D., Surg.-Gen. Ind. Med. Dept., Sanitary Commissioner of Govt. of India.
C. 1860. Curtis, Josiah, Boston, U.S.A.
N.R. 1876. Day, E. J., Dorchester.
N.R. 1883. De Renzy, A.C.C., C.B., Surg.-Gen. Ind. Med. Dept. (*retired*), 9, Sydenham Villas, Bray, Co. Wicklow, Ireland.
C. 1878. Delmas, Louis H., M.D., P.O. Box 524, Havana, Cuba.
C. 1853. Dempster, Charles Carroll, M.D., Dep. Insp.-Gen. (*retired*).
N.R. 1852. Dickinson, Wm. Lindow (address unknown)
C. 1867. Dickson, E. Dalziel, M.D., Physician to British Embassy, Constantinople.
N.R. 1871. Domenichetti, Richard, M.D., Trinity Lodge, Louth, Lincolnshire.
N.R. 1864. Donnet, J. J. L., M.D., R.N., 5, Park Road, Bognor, Sussex.
N.R. 1876. Downes, A. H. J., M.D., Chelmsford, Essex.
N.R. 1863. Ducket, C. A., M.D., 12, St George's Terrace, Great Yarmouth.
N.R. 1871. Dunlop, James, M.D., 18, Carlton Place, Glasgow, N.B.
N.R. 1871. Dyke, Thomas Jones, Merthyr Tydvil, South Wales.
N.R. 1882. Fasken, William A. D., M.D., Surg. Ind. Med. Dept., Messrs. Grindlay and Co., 55, Parliament Street, S.W.
N.R. 1875. Fox, J. Makinson, M.D., The Grove, Lymm, Cheshire.
N.R. 1883. Furnell, M. C., M.D., Surg.-Gen., Shearman's Gardens, Madras.
C. 1851. Galland, —, M.D., Malta.

Elected.

C. 1863. Hall, E. Swarbreck, 58, Campbell Street, Hobart Town, Tasmania.
N.R. 1852. Hardwicke, Junius, Clifton Lodge, Rotherham.
N.R. 1876. Hardwicke, H. Junius, M.D., Burton Lodge, Wostenholm Road, Sheffield.
N.R. 1872. Harris, Henry, M.D., Trengweath, Redruth, Cornwall.
C. 1852. Harrison, J. B., M.D., Higher Broughton, Manchester.
N.R. 1880. Harrison, R. Charlton, M.D., 13, Sandringham Gardens, Ealing, W.
N.R. 1855. Haviland, Alfred (address unknown).
C. 1852. Hebra, Ferdinand, M.D., Vienna.
N.R. 1881. Henderson, G. C., M.D., Kingston, Jamaica.
N.R. 1882. Herring, John F., Builth, South Wales.
N.R. 1875. Hime, T. W., M.D., Bradford, Yorkshire.
C. 1853. Hirsch, August, M.D., Berlin.
N.R. 1871. Hogg, F. R., M.D., R.H.A. (address unknown).
N.R. 1875. Home, Sir Anthony D., K.C.B., V.C., M.D., Surg.-Gen., India.
N.R. 1866. Home, A. H., M.D., Hullerhirst, Steventon, Ayrshire.
C. 1851. Hoskins, S. E., Guernsey.
N.R. 1881. Irwin, Chamney Graves, M.D., Dep.-Surg.-Gen., Edinburgh, N.B.
N.R. 1855. Jacobson, T. E., Sleaford, Lincolnshire.
C. 1886. Janssen, —, M.D., Inspecteur en Chef d'Hygiène, Brussels.
C. 1851. Kellay, —, M.D., Beyrout, Syria.
N.R. 1883. Kirker, Gilbert, R.N., M.D., R.N. Hosp., Gosport.
C. 1857. Leas, Jose Fernandes da Silva, Cape de Verde.
C. 1875. Lent, —, M.D., Cologne.
C. 1886. Le Roi de Méricourt, L., M.D., 5, Rue Cambacères, Paris.
N.R. 1879. Lilburne, James, M.D., Dep. Inspec.-Gen., Duncrivie House, Milnathort, N.B.
C. 1851. Litt, W., Veterinary Surgeon, Shrewsbury, Salop.
N.R. 1873. Little, James, M.D., 14, Stephen's Green North, Dublin.
C. 1857. Lowtzoff, —, St. Petersburg, Russia.
N.R. 1881. Lyons, R. T., M.D., Surgeon-Major, Rampart Barracks, Fort William, Calcutta.
C. 1870. Mallet, —, M.D., Warsaw.
N.R. 1865. Mapother, E. D., M.D., 6, Merrion Square North, Dublin.
N.R. 1867. Marshall, John, 13, Liverpool Street, Dover.
N.R. 1860. May, George, jun., Reading, Berks.
C. 1854. Moffatt, Thomas, M.D. (address unknown).
C. 1886. Moleschott, Jacob, M.D., Rome.
N.R. 1872. Moore, John Wm., M.D., 40, Fitzwilliam Square West, Dublin.
N.R. 1884. Moore, W. J., C.I.E., Surg.-Gen. (address not known).
C. 1851. Monzon, Rafael, Barbacaos, New Grenada.
C. 1853. Neumann, —, M.D., Berlin.
C. 1851. Noble, Daniel, M.D., 258, Oxford Road, Manchester.
N.R. 1881. Oldham, Chas., Surg.-Major Ind. Med. Dept., Shurdington Road, Cheltenham.
C. 1872. Poggio, Don Ramon Hernandez, Cuba.
C. 1854. Purple, S. S., M.D., New York, U.S.A.
N.R. 1879. Ransom, W. H., M.D., F.R.S., Nottingham.
N.R. 1875. Ransome, Arthur, M.D., F.R.S., Devisdale, Bowdon, Cheshire.
N.R. 1884. Ray, George, Surg.-Gen., 76, Jermyn Street, S.W.
N.R. 1874. Reid, Walter, M.D., Staff-Surgeon, Royal Naval Hospital, Haslar.

Elected.

N.R. 1866. Robertson, James, M.D., Alfred Place, Melbourne.
C. 1886. Rochard, G., M.D., Paris.
N.R. 1863. Rowe, T. Smith, M.D., Cecil Street, Margate.
N.R. 1879. Ross, J. T. C., C.I.E., Surgeon-General Ind. Med. Dept., The Grove, Ryde, Isle of Wight.
N.R. 1881. Russell, E. G., M.B., B.Sc., Surg. Ind. Med. Dept., Gauhàti, Assam, India.
N.R. 1875. Russell, J. B., M.D., 1, Montrose Street, Glasgow.
N.R. 1859. Sanderson, J. Burdon, M.D., F.R.S., 50, Banbury Road, Oxford.
C. 1851. Sankey, F. F., Malta.
C. 1879. Sarrell, Richard, M.D., Constantinople.
C. 1873. Schleisner, —, M.D., Copenhagen.
C. 1874. Schlimmel, —, M.D., Teheran.
C. 1852. Schürmayer, Professor, Heidelberg, Germany.
N.R. 1858. Scratchly, G., New Orleans, U.S.A.
C. 1851. Sharpe, Richard, Beverley Road, Hull.
N.R. 1869. Shaw, James, Insp.-Gen. (address not known).
C. 1853. Sigmund, C. L., Vienna.
N.R. 1867. Smith, R. Wagstaff, Mount Rundell, Harborne, Birmingham.
C. 1886. Sonsino, Prospero, 11, Via San Lorenzo, Pisa.
N.R. 1854. Spinks, C. N., Sankey Street, Warrington.
C. 1851. Spooner, W. C., Vet. Surg., Southampton.
C. 1876. Sternberg, Geo. M., M.D., United States Army, Port Point, San Jose, San Francisco, California.
N.R. 1885. Sturridge, P. F., Kendal, Westmorland.
N.R. 1880. Tatham, John, M.D., Town Hall, Salford, Manchester.
C. 1860. Taylor, H. Sharp, 15, Quarry Street, Guildford.
C. 1855. Tholozan, J. Désiré, M.D., Teheran.
N.R. 1853. Todd, J. George, Evenwood, West Auckland, Durham.
N.R. 1885. Tomes, Arthur, Surg. Ind. Med. Service, Bengal.
N.R. 1873. Townsend, Stephen Chapman, C.B., Surg.-Gen. Ind. Med. Dept., Sanitary Commissioner for Central India.
C. 1851. Tribes, E., Nismes, France.
C. 1855. Tribuchet, Adolphe, Paris.
C. 1853. Valerj, —, M.D., Rome.
C. 1855. Virchow, Rudolf, Professor, 10, Schellings Strasse, Berlin.
C. 1856. Von Iffland, —, Beaufort, New Quebec.
N.R. 1879. Watson, Alexander, M.D., R.N., Dep. Insp.-Gen., Army and Navy Club.
C. 1853. Watson, John, M.D., Jamaica.
N.R. 1880. Whishaw, J. C., M.D., Surg.-Major Ind. Med. Dept., Lucknow.
N.R. 1880. White, J. Berry, M.D., Surg.-Major Ind. Med. Dept., address not known.
C. 1861. Whitelaw, Wm., M.D., Kirkintilloch, Dumbartonshire.
N.R. 1854. Wiblin, John, Southampton.
N.R. 1865. Williams, C., 48, Prince of Wales's Road, Norwich.
N.R. 1884. Williamson, R. E., M.B., Lymm, Cheshire.
N.R. 1872. Woodall, John (address not known).
N.R. 1875. Wortabet, John, M.D., St. John's Hospital, Beyrout.

TRANSACTIONS
OF THE
Epidemiological Society of London.

A REVIEW OF THE PAST YEAR,

WITH SOME

REMINISCENCES OF CHOLERA AND TROPICAL FEVERS.

By WALTER DICKSON, M.D., R.N., President.

Inaugural Address of Session 1886–87.

(*November 10th*, 1886.)

Gentlemen,—It is my first duty to tender you my warmest thanks for the distinction you have been pleased to confer on me by electing me for the second time to preside over your deliberations in this place. Be assured I shall ever prize it as a high honour to have obtained your approval, and will endeavour, to the best of my ability, to perform the duties and maintain the traditions of the office to which you have advanced me.

I have to congratulate the Society on its present flourishing condition. The number of our members has increased to 217, and of our honorary members to seven, and includes many of the most distinguished epidemiologists at home and abroad. I can also congratulate the Society on the amount and interest of the work done during the last session. Notwithstanding the deterrent effect of an unusually cold and prolonged winter, the meetings were well attended, and several of the most important topics of epidemiological science were brought before us in papers of great value and interest, and in the profitable discussions that followed. Of such I may enumerate, Cholera as it has appeared Epidemically in our Ships and Fleets, and its Prevention by Restrictive Measures; Small-pox and the great Results of Compulsory Revaccination in Germany, together with the alleged Injuries therefrom; Quarantine, and its Discussion at the International Sanitary Conference at Rome last year; Hydrophobia Statistically Considered in Relation to its

remarkable Increase in England of late years; Diphtheria as a Chronic and Rejuvenescent Disease; and Scarlet Fever, more especially in its newly-discovered Connection with a local Disease affecting the Udder of the Cow, which last gave rise to a very important contribution, and excited great interest, not only in our own medical ranks, but among veterinary practitioners and gentlemen connected with agriculture and the milk-supply of the metropolis, several of whom we were glad to welcome among us, to share in our proceedings, and give us the advantage of their high intelligence and practical knowledge of domestic animals and their diseases.

Some of the subjects to which I have referred are likely, we trust, to be treated continuously by the authors and speakers to whom we have been indebted, and from whom we may hope for further contributions. I trust also that we shall have from our corresponding and foreign members original papers on local and climatic peculiarities manifested in the epidemic diseases of their various countries. We have lately received from Dr. Corney, of Fiji, an account of a remarkable outbreak of cerebro-spinal fever among the Polynesian coolies in those islands. I have recently been in correspondence with a physician of Buenos Ayres, Dr. Perreira Rego Filho, on the subject of tetanus, wherein he is making widespread inquiries, but on which I have been unable to give him the copious statistical information he desired, by reason of the comparative rarity and unimportance in this country of that formidable disease, which specially abounds in hot climates and among coloured populations, and with which our Indian and Colonial brethren are likely to be far more conversant than British practitioners. I mention the circumstance mainly as a pleasing evidence of the appreciation in which the Society and its work are held in regions so remote as South America and the islands of the Western Pacific.

We gladly welcome among us those members who have been elected by the Society during last session, and represent various departments of public medicine in the national services and civil life. We confidently expect their valuable co-operation in our proceedings, and that they will help to maintain the worthy traditions of the Society in focussing, as it were, the light they have derived from their wide and manifold experience of disease at home and abroad, for the benefit of their fellow-members and the public.

We have to deplore, since our last opening meeting, some heavy losses. Dr. Gavin Milroy had taken the warmest

interest in the Society from the beginning, had filled the offices of Secretary, of President for two years, and of Vice-President. He was universally known as an ardent epidemiologist. Profoundly learned in the literature of the subject, and practically conversant, through his public missions to the Crimea and the West Indies, with some of the most important diseases of armies and civil populations, he devoted the energies of his life, prolonged in peace to eighty years, to the great questions of sanitation and the prevention of disease. He had the gratification to find the views he early entertained, and the reforms he advocated, fully recognised, and in many instances adopted, within the thirty-five years that embraced his professional labours and writings in this connection. In this Society and by his private friends he was highly esteemed for kindness and courtesy, as well as ability, and his disinterested devotion to science and simplicity of life rendered him the very type of a medical philosopher. Until disabled by age and infirmity, Dr. Milroy was indefatigable in his attendance at our meetings, and to the last took a lively interest in our doings. To mark his sense of the importance of his favourite studies, he has endowed a lectureship at the Royal College of Physicians, which will serve to perpetuate his name and work to succeeding generations.

Another eminent colleague has been removed from among us by untimely death at the age of fifty-seven. Dr. Thomas Spencer Cobbold was for several years one of our most distinguished members, and had enriched our *Transactions* with some valuable memoirs on Filariæ and other Parasites. In his special department of Helminthology he had acquired a world-wide reputation for careful observation and thoughtful research, and held a high position as a cultivator of natural science in many branches. He was early elected a fellow of the Royal and other Societies, in recognition of the value and originality of his writings on biology, which were very numerous. Dr. Cobbold possessed the rare advantage of combining extensive and minute knowledge with an attractive, natural eloquence, and power of lucid exposition, which gained equal acceptance with learned and popular audiences. When Swiney Professor of Geology, his lectures were attended by crowds of intelligent listeners, to whom he imparted no small portion of his own enthusiasm for scientific truth. Dr. Cobbold was not only remarkable for his high attainments, but for his social qualities. Singularly disinterested, genial and bright, a most accomplished gentleman, his memory will always be cherished by those who had the

happiness of his friendship, while his eminent services to science will long be held in respectful remembrance.

We have also to record the death of one of our oldest corresponding members (elected in 1852), Dr. George Varentrapp of Frankfort, who practised for more than fifty years, and filled a high position as Medical Officer in that city. He was deservedly held in the highest esteem and regard for his unwearied, unselfish exertions in the cause of science and humanity. As a sanitarian and philanthropist, and a leader in every good work during a long and useful life, his fellow-citizens celebrated five years ago his Doctor's jubilee (that is, the fiftieth anniversary of his taking his degree) with much enthusiasm, and dedicated, for his special acceptance, a volume relating to the hygienic and charitable organisation of Frankfort, in which he had had so great a share, accompanied by an eloquent and touching address, expressive of their admiration of his character, and gratitude for his services.

On the last occasion I had the honour to address you, some recent epidemics at home and abroad afforded several points of interest for consideration. A violent and widely diffused outbreak of cholera in Spain had just run its course, and had terminated on the approach of cool weather, after killing 120,000 of the population. The total exemption of the neighbouring and closely connected kingdom of Portugal was a noteworthy and not easily explicable feature of that formidable epidemic. No recrudescence of the scourge is recorded to have occurred in the Spanish peninsula in the past summer, but the kingdom of Italy had continued to suffer from time to time in various localities, although in a less degree than in the former epidemic of 1884, and chiefly by reason of the exemption of Rome, Naples, and other large cities. A few cases in various districts had occurred in the winter, but epidemically it first appeared in the spring of this year, almost simultaneously at Brindisi and Venice, and since April many other cities and villages on or near the Adriatic coast have been affected. In Venice, in the first week of June, 215 cases of cholera were recorded, with 121 deaths. In the Venetian province, the inland cities of Padua, Verona, and Vicenza suffered, but in less degree; and subsequently, by its extension into Istria, the Austrian ports of Trieste and Fiume yielded some cases. In the large maritime province of Apulia, in addition to the commercial *entrepôt* of Brindisi, Bari, Barletta, Taranto, and other seaport towns experienced considerable losses, and further north, in the Emilian province, Ravenna, Ferrara, and Bologna. In the valley of the Po it was contended, and two English

physicians are said to have supported the view, that the disease affecting that district was not true cholera, but a severe form of typhoid, with a mortality high indeed, but greatly less than is normally found in the Asiatic type of disease.

On the south-west coast of Italy cholera has made itself felt at Torre dell' Annunziata, Castellamare, and other places near Naples, but not in that great city itself, which suffered so much in the former epidemic. Lately it is stated to have visited Cagliari, the capital of the island of Sardinia.

In September, also, beyond the confines of Italy, cholera was reported to have appeared on the Danube, and in Buda-Pesth and Szegedin to have recently assumed epidemic proportions. With its tendency to propagate itself along rivers, fears were entertained of a great extension of the disease in that part of Europe, but the considerable fall of temperature that occurs at this season in Hungary and the neighbouring countries appears to have checked it.

A minor outbreak of cholera in France, limited to a part of Brittany and to the months from October to February, serves to remind us that, as in its first visitation in this country, in November 1831, a high temperature is not invariably, although doubtless very frequently, an important factor in the development of the disease. Different accounts are given of its origin on the secluded coast of Finisterre, in a cluster of small fishing towns away from the great channels of intercourse and traffic. Dr. Thorne Thorne states that the disease was apparently introduced into Concarneau, the town first invaded, from Toulon by the arrival of seamen on furlough from war-ships or transports recently returned from Tonquin. Other accounts attribute the outbreak to importation from Brest, and regard it as part of the declining epidemic that afflicted France in 1884-85. The Minister of Commerce, who discharges also the functions of Minister of Health, sent a commission of investigation, whose report was published by the Academy of Medicine in February last. Doctors Proust and Charrin inquired minutely into all the circumstances attending the appearance of the malady at Concarneau, a small port with 5,000 inhabitants, where it carried off thirty-five persons, and traced it thence to the neighbouring coast towns in that remote corner of France. They showed that, in several instances, it was conveyed by sick persons or their property, or by those who had been much in contact with the sick. Audierne and Guiloinéc suffered most; the former, out of a population of 1,700, had 400 cases and 144 deaths; the latter, with 2,000 inhabitants, had seventy-one deaths. The very poor, and those who indulged in alcohol,—

no uncommon vice of Breton fishermen,—were chiefly victims, and the deplorably defective sanitation of all these places as to water and everything else doubtless helped greatly to intensify the disease. It was observed that those who, while watching the sick, took their meals in the bedchamber seemed especially liable. No premonitory diarrhœa is said to have occurred, though afterwards minor bowel-complaints co-existed with the prevalence of the cholera during the winter months. The smaller towns suffered most; Brest and Douarnenez in a less degree; and the only inland town affected, and that not severely (thirty-five deaths in a population of 15,000), was Quimper, the chief town of the Department, which has necessarily much communication with all the others. The troops were not affected, and the commissioners —rightly, no doubt—ascribe this exemption to their superior sanitary surroundings, and the prophylactic measures that were diligently carried out in their behalf. The report is chiefly interesting from the minute care with which the spread of the epidemic is traced from town to town through individuals or their belongings. Drs. Proust and Charrin consider it probable that the disease was imported into Concarneau by fishermen from Spain; but they sternly rebuke the many sanitary shortcomings of those towns, and freely expose the culpable neglect which inflicted on them so heavy a mortality.

From its first appearing in commercial cities on the Adriatic, it was inferred by many that this year's Italian epidemic was probably imported. Brindisi, in particular, is in constant communication with the East by sea, and has relation by railway with many coast towns. These ancient cities, generally massively built of stone, to last for centuries, abound in insanitary conditions of every kind; and the poorer inhabitants, living in the summer months almost exclusively on uncooked vegetable food, melons, salad, and the like, and drinking freely of water from, in many instances, impure and tainted sources, are always prone to bowel-disorders. The malady has therefore a highly favourable soil for its propagation and diffusion during the six months of almost tropical temperature, amid a population crowded in walled towns and villages, and ignorant or careless of the most obvious elementary laws of health. Even in the wealthiest families there is much room for improvement. An English lady who resided among them has lately recorded her experience that where, as often happens, there is an enormous store of domestic linen, the necessary operation of washing it is performed only once a year, with much ceremony, and assumes

the importance of a great annual function. The primitive filthy state in which the poorer classes, otherwise estimable persons, live, is stated to be beyond belief; and to equal, if not excel, an instance lately reported to have occurred here in London, of an old lady who refused to become an inmate of Chelsea workhouse, on the ground that she would be compelled to use a bath, saying that she had had one ten years ago, and was determined never to submit to take another. Many of our poorer classes are equally conservative in clinging to habitations of proved unwholesomeness even when better ones are available. It has been recently stated, on good authority, that in pulling down bad buildings and erecting others on their site, for the benefit of the working-classes of London, the Metropolitan Board of Works had spent a million and a half. Yet great difficulty has been experienced in inducing the more squalid and miserable of our population to enter into those improved dwellings, as they preferred, when turned out of the old, to migrate to other dens of filth and wretchedness, where the surroundings would be more familiar and congenial to their cherished ideas as to comfort.

The Italian people, with few enlightened exceptions, do not realise, as yet, the paramount value of sanitation, and are firm believers in the foreign origin of cholera, that it is always imported, and chiefly to be prevented by maintaining rigid quarantine regulations. "Dirt", they say, "we have always with us, but cholera seldom." Both at Brindisi and Venice the introduction of the epidemic was ascribed to passengers from the East or to their personal effects, especially foul linen; and at Palermo, where a fatal epidemic raged in September and October 1884, and carried off 3,000 persons, it was confidently asserted that the linen of the captain of a vessel from an infected port, who had suffered from cholera, was the medium through which the morbid poison was conveyed into the city and radiated from the laundress's house, where the first cases occurred, over the whole population with destructive violence. Hence the relaxation of the old quarantine laws was deeply resented by the people, who clamoured for their reimposition, although well aware of the injury thereby caused to the commercial prosperity of their port. The central Government at Rome found themselves compelled to yield to some extent to the popular voice. The truth is that in these Southern lands, where sanitary science is all but unknown, it is only enlightened statesmen, or men who have travelled in and thought over the better condition of other countries, who are fully alive to the absurdity of the old restrictive system, and to the necessity for local sanitation,

and the incomparable advantages to be derived from it. We may therefore heartily congratulate our distinguished colleagues, who so worthily represented the epidemiological science of this country at Rome, on converting to their views so many members of the International Conference, as to lead to what, only a few years ago, would have been considered a triumphant result, namely, the abolition of land quarantine and the reduction of the period of the detention of ships from infected countries to five days.

There is every reason to expect that in no long time this period will be still further lessened, until the embargo on intercourse with ships become little more stringent than in this country. At the same time it should not be forgotten that we are nearly half a century in advance of these States in domestic sanitation, which, from the structure of their towns, the heat and dryness of their climate, and consequent frequent scarcity of water, and also from the inveterate unwholesome habits and customs of their inhabitants, would in most localities be a far more difficult and expensive undertaking than with us. They do not possess the vast accumulation of capital, or enjoy the commercial tribute of the whole world, which have fallen to the lot of this happy country, nor have they the physical advantages of climate which are so eminently conducive to the health and comfort of our city life. It is not to be wondered at, therefore, that they cling to the primitive rough and ready method of closing the door to the aggressor, and, at some sacrifice of their material prosperity, endeavour to avert the invasion of pestilence by the same simple means which for many generations they had found tolerably effectual in excluding the plague, malignant typhus, and other forms of contagious disease, whose etiology and mode of communication are less obscure, complex, and mysterious than those of cholera.

While, therefore, the long detention adopted by the Conference appears to be unnecessary and vexatious, and is sure to be curtailed in deference to progressive enlightenment, a short period in which to ascertain the truth as to the sanitary condition of a suspected vessel, often wilfully concealed or misrepresented, is, I venture to think, not only justifiable, but in certain circumstances desirable, even in this country, and *a fortiori* in those regions nearer the Oriental home of the disease. When, for example, cholera exists on the Continent of Europe in our immediate vicinity,—say, in the ports on the coast betwixt the Elbe and the Loire,—all preventive precautions to exclude it by quarantine regulations would be idle and useless, so many ways of access being

open; but in the case of a vessel some few days from an infected port, say from the Baltic, Peninsula, or Mediterranean, caution is advisable before admitting her to free communication. A legitimate detention for observation and inquiry, for medical inspection, removal and isolation of the sick, if properly conducted, need not (to preserve the ancient *quaranta* tradition) exceed the maximum of forty *hours*,—no very great sacrifice to make in the interest of the public safety.

For the rest, as Dr. Thorne assured us in the admirable paper with which he lately favoured us, recounting the proceedings and arguments of the Roman Conference, we should be well content with the general and hearty recognition accorded by all to the great results of English sanitation in often preventing and always mitigating epidemic disease. This nation owes a deep debt of gratitude to the Medical Officers of the Local Government Board, for their strenuous and successful exertions in discovering our sanitary defects, not always confined to cities, and pointing out the means of correction. Their eminent chief, who has done so much for preventive medicine by thoughtful organisation and indefatigable labour, has, in a recent official publication, summed up our position in words so pregnant with salutary wisdom, that I cannot forbear quoting them:—"For European countries which have secured their soil, water, and air against befoulment, there is little or no danger of cholera, no matter though the disease be actually brought into their midst, whereas communities which have not obtained this result encounter serious risks from cholera when it chances to be introduced among them. England trusts in pure earth, water, and air, and regards such purity as sufficient to prevent the spread of cholera in a European community." And again, "Quarantine results rather in hazardous concealments and evasions than in any effectual exclusion of cholera." Those who repose their trust entirely on such regulations, delude themselves into a false security, which encourages them to omit the great prophylactic measures which our experience in 1849, 1854, and 1866 have amply demonstrated to be alone reliable against the spread of the disease. Contaminated water, since Dr. Snow's memorable researches, has ever been the object of greatest solicitude, as apparently the prime factor in disseminating the morbid poison, germ, microbe, or whatever it may be, and continued microscopic investigation into its nature is eminently desirable. Dr. Buchanan probably expresses the present state of opinion of epidemiologists in this country when he writes that, "although

Dr. Koch has not succeeded in determining the specific relation of cholera to comma bacilli in tank-water, and has formed his conclusions too hastily, students of the subject should not be too hastily turned away from the further pursuit of researches in the same direction."

Similar remarks might in some degree apply to the labours of another eminent scientist, whose efforts to avert a rarer, but even more terrible and mysterious disease, have been watched with intense interest, and hailed everywhere with grateful recognition. The recent formidable extension of hydrophobia has afforded M. Pasteur the opportunity of trying his prophylactic system of inoculation with attenuated virus on a large scale, and under favourable circumstances. He has not had to contend with the irrational obstruction and hindrances to scientific research which, in England, have been imposed on us through inconsiderate short-sighted legislation, but has received, as was his due, encouragement and support from all quarters. M. Pasteur's experiments seemed at first remarkably successful, but of late a considerable number of disappointments have shown that his system is still on its trial, and that it would be premature to form a conclusive judgment upon its merits. Yet any means preventive of a cruel and incurable disease, devised in a true philosophical spirit by one whose achievements in kindred investigations have been brilliantly successful, must be accepted with respect and gratitude, and hopefulness that, with further experience, his method of neutralising the poison of hydrophobia may be further developed and improved, and bear even better results. The practical outcome of the interest and anxiety felt on rabies and its consequences, has in London been chiefly beneficial by leading to the destruction of many useless, ownerless dogs, and to the more efficient control of the survivors. More attention should be paid to the fact that many suspected dogs are not rabid, and that, in the interest of those bitten by them, it is unwise to destroy them immediately. Should the dog, probably only ill-tempered or suffering from curable disease, survive a fortnight's detention and watching, much anxiety would be spared to the persons bitten.

This time last year I took occasion to allude to the remarkable endemic exanthem known as Verruga, or bleeding wart of Peru, which has existed from remote antiquity in the deep, unwholesome ravines of the Andes, between 9° and 16° south latitude, at an elevation of 3,000 to 8,000 feet above the sea. The disease appears to be confined to this locality, and has proved very fatal to foreigners temporarily residing there.

Some fresh light has been thrown on its pathology recently through the melancholy result of an experiment made by a promising young Peruvian physician, Senhor Daniel Carron. He inoculated himself in September 1885 with the blood of a patient suffering from verruga in the hospital at Lima. After twenty-three days of incubation he produced on his body some of the chief symptoms of the disease—adynamic pyrexia and general dermatitis—and died on the thirty-eighth day. What has been called the fever of Oroya, a fatal asthenic fever infesting the same locality, is now considered by some of the Lima physicians as part of the same disease, but sometimes carrying off the patient before the characteristic eruption of bleeding warts has had time to appear. Professor Izquierdo, of Santiago in Chili, is reported to have discovered the germ of verruga in a peculiar bacillus, filling the arterial and venous capillaries of the skin and subcutaneous tissue, circulating in the blood, and provoking by irritation the genesis of the local morbid changes. Verruga is therefore considered to be a zymotic malady, with a considerable period of incubation, probably, like paludal fevers, of telluric origin, and possibly dependent on the introduction into the body of a peculiar microbe, causing complete disorganisation of the blood. Although not necessarily contagious, it has been proved to be inoculable, through the sad history of Senhor Carron's experiment.

I have derived this information from M. Rey, in that valuable periodical, the *Archives de Médecine Navale*, which, under the able editorship of our distinguished colleague and corresponding member, M. le Roy de Méricourt, communicates much interesting knowledge, and sheds lustre on the French Navy. Their mutual familiarity with other Latin nations and tongues gives great facility to our naval brethren of France for acquiring information in some little known regions about diseases almost new to us, and enables them to spread abroad in monthly publications the thoughtful labours of foreign physicians, in addition to their own valuable maritime experience.

The last report on the health of our own Navy, viz., that of 1884, is of great interest, chiefly in relation to the severe forms of enteric and other fevers that afflicted the considerable force employed in North-east Africa during the recent war. The sickness ratio of the total force all over the globe from continued and paroxysmal fevers in that year was thereby raised to 100 cases per 1,000 men from the average of the ten preceding years of 64 cases per 1,000 men. The mortality rate from those diseases, in like manner, rose to 2

per 1,000, and the invaliding rate to 8.25 per 1,000, instead of the annual mean ratios of 1 per 1,000 and 2.4 per 1,000. The accounts from Souakim, Massowah, and other places are replete with interest in regard to the etiology of those diseases as they fell on our seamen and marines afloat and on shore in that calamitous expedition. Ships lying in Malta harbour also suffered much from fever, which was ascribed partly to the vast deposit of filth that had been allowed to accumulate in a tideless basin, and generated most offensive emanations, and partly to the extensive excavations and upturning of soil involved in constructing the works for improving the drainage of Valetta.

On the West Coast of Africa the paroxysmal fevers appear to have been of a mild type. Although the ratio of number of cases in the squadron was nearly double the average, 200 per 1,000, compared with 111 per 1,000, the invaliding was only 9 per 1,000, and no death whatever is recorded. One vessel spent four weeks in September and October up the Niger, and after an incubation period of from three to six weeks, the whole white crew of seventy men were disabled by malarious fever, except six persons. No death or invaliding had occurred at the date of the report, but the anæmia and debility resulting from such attacks often lead to invaliding some time afterwards.

Only one case of yellow-fever seems to have occurred in the whole Naval force during the year. This was at Callao, on the Pacific station, where a man, who had slept three successive nights on shore, was attacked three days afterwards, and died on the fourth day of the disease, with albuminous urine and dark-coloured ejecta. The shore is here marshy, and paroxysmal malarious fever is endemic. There was no yellow-fever epidemic in the place, though sporadic cases are said to be occasionally met with.

The incidence of cholera has this year been also very light. Five cases are reported, all at Amoy, in China, with four deaths. There was a cholera epidemic on shore, and much diarrhœa on board. By energetic measures of prophylaxis and disinfection, and timely treatment, the outbreak was soon quelled. One of the reputed causes of the disease in this instance was the free use of aerated water, manufactured in the town from sewage polluted water—a frequent source of danger, doubtless, even in this and other countries where those beverages are so largely consumed. A considerable improvement has taken place in the health of the Naval force in China of late years, due, I think, to the vessels being sent oftener to Japan and the cooler parts of the station,

and thereby escaping the pestilential heats of summer, so fatal to our countrymen on the coast of Southern and Central China.

In my service there of two years, as flag-surgeon in the last war, in immediate charge of about 1,000 men, intestinal disorders constituted 25 per cent. of the whole amount of incapacitating illness, 70 per cent. of the whole mortality, and 40 per cent. of the invaliding. In thirty months of 1859-60, the cases of dysentery were 185 in number, of which 17 died and 30 were invalided; while of diarrhœa there were 606 cases of considerable severity. Cholera was rare: only four cases, all contracted on shore, and not spreading on board. I attributed much of this bowel-disorder to the use of water got from the shore,—the wholesome practice which had hitherto obtained, of the crew drinking only water distilled on board, having been discontinued through a misjudging spirit of economy. That the mode of living had a great share in causing those maladies, was substantially proved by the fact that none of the numerous officers, though exposed to the same climatic conditions, were seriously affected.

Venereal diseases in the Naval Report of 1884 show a portentous increase, more especially on the Home station, where the ratio of cases of syphilis is double that of the ten preceding years, and the daily number thereby incapacitated for duty has risen to 11 per 1,000. If minor kindred disorders be included, the number daily sick from this cause has been 20 per 1,000, which, when compared with the average ratio of the previous ten years of 11 per 1,000, points to the conclusion that the recent removal of legislative restrictive measures has grievously impaired the health of our seamen in the Home ports. It is some compensation that in nearly all the foreign stations, particularly those where prophylactic regulations are enforced, there is a considerable diminution of venereal disease. Yet in the whole force the absence from duty caused by it is very great, showing an aggregate in the year of 222,000 days, or five days per man, which is equal to that caused by all the zymotic, rheumatic, respiratory, and digestive diseases put together, and higher than in any year since the reports began in 1856. This startling fact would appear to indicate the necessity of increased stringency, rather than the withdrawal of the salutary preventive means that experience has shown to be so beneficial. In this connection, Fleet-Surgeon Hadlow has reported a recent remarkable change of opinion at the Cape of Good Hope, where the repeal of the Contagious Diseases Acts in the Colony had been followed by such disastrous consequences, through im-

migrants, on their way to the diamond-fields and gold-diggings, disseminating those loathsome diseases so widely among the community, that it had become necessary to reimpose the restrictive measures that had imprudently been abandoned.

In this Society a perennial interest attaches to the subjects of cholera and tropical fever. I will therefore ask your permission to avail myself of the short time that remains at my disposal in briefly recounting the history of a few outbreaks of those diseases that occurred in my naval experience. They were not extensive or prolonged, and in the microcosm of a ship the more noteworthy points of their etiology could be more definitely and fully ascertained than in a less limited field of inquiry.

The first I shall allude to befel H.M.S. *Archer*, a corvette, with a crew of 200 men, in the Baltic in 1854, the first year of the Russian war. She had left England in March, and arrived at Stockholm on the 6th of April. Like almost all the vessels of that fleet, she had been fitted out with the utmost despatch, manned with a wretched crew, many of them landsmen, picked up in rags on the streets of London, and, such was the exigency of the time, sent to sea badly provided with clothes, bedding, and other comforts essential to the health and well-being of seamen. The food alone was satisfactory, but even that was of quality and quantity to which they were unaccustomed, often gave rise to indigestion, and, combined with a cold, late spring, to boils, whitlows, and other cutaneous affections. Many of the crew suffered from the usual effects of exposure, pulmonary affections, rheumatism, and the like, and the sick-list averaged 12 per cent. It was some weeks before the undisciplined horde that had been so hastily got together acquired any semblance to the orderly crew of a man-of-war. The ship was at once employed on arduous service, having to keep the sea in all weathers, blockading the enemy's ports and destroying his commerce. Such cruising on the coast of Courland was rendered more trying by the cold, raw, tempestuous climate of the prolonged spring of that year. Matters improved during the few weeks of summer; but September brought back the winds, rain, and constantly wet decks, inevitable when a vessel is very rarely at anchor, but ever on the move, whatever might be the circumstances. The hatches and ports were generally fastened down in the south-west gales that prevailed, excluding light and air to a great extent, but not water.

Such was the damp, close, insanitary condition of the ship when, on the 23rd of September, off Memel, in East Prussia, she captured fifteen small Russian vessels laden

with grain, which were stealing along the coast to dispose of their cargo at a neutral port. These prizes were sent into Memel in charge of 69 men, and forthwith a heavy gale springing up, compelled the ship to stand off the land under steam, and she was unable to return to pick up the prize-crews till four days afterwards. Meanwhile, those men had been badly lodged, sleeping on bags of damp grain, ill-fed, and, straggling into the town, had indulged in alcoholic excesses and other irregularities. The weather was cold and wet. Memel was reported to be healthy, but several cases of cholera, which then was epidemic in many parts of Europe, had occurred during the summer. On the 28th of September the men returned on board the ship, but on the next day one was found lying on the deck, with vomiting and purging, severe cramps, and all the symptoms of the collapse stage of cholera. He had had diarrhœa for some hours, but thought so lightly of it that he had continued to perform his duties as captain of the hold in cleaning and whitewashing. He died in fifteen hours. On the same day, the 30th, a marine was brought off from the port, who had, while intoxicated, wandered about the streets in a miserable plight for some days without food or shelter. He had vomiting, rice-water purging, and cramps, but did not show the livid and shrivelled skin of collapse. He survived till the 7th of October, and the stools having improved and urine been secreted, hopes were entertained of his recovery; but symptoms of typhus supervened, with delirium, stupor and asthenia, to which he soon succumbed. Several cases of diarrhœa, more or less severe, occurred among the men of the prize-crews, and also a few among the ship's company who had not been on shore. A marine, who had been all along on board, and had had no direct contact with the cholera-patients, suffered from diarrhœa, at first slight, but soon developing into cholera, though without cramps or intense collapse. He improved for a time, but this amendment was delusive, for asthenic fever appeared on the seventh day, and on the tenth day he expired, after vomiting a small quantity of black fluid and discharging a very copious rice-water evacuation. In all, forty cases of severe bowel-complaint occurred in October, besides many others not severe enough to require withdrawal from duty. The great majority of the worst cases were in men who had been on shore at Memel, although they formed only one-third of the ship's company. In the first two days of the epidemic thirteen cases were of men who had landed, and only two of those who had remained on board. A better hygienic condition of the ship would probably have still further increased this difference.

The only other instances of cholera I shall bring before you occurred at Aden, in Arabia, in March 1859, to some of the crew of H.M.S. *Chesapeake*, the flag-ship on the East India station. She had been in commission for some time, having been despatched to Calcutta in 1857, to aid in the suppression of the mutiny; had spent the early part of 1858, and also the hot months of May and June, in the Hooghly, and had at intervals visited Madras and Ceylon, where the crew had been employed for some weeks in very arduous duty, recovering Government treasure from the wreck of a steam-packet which had been lost on the coast of that island. The ship had subsequently gone to the Red Sea, in consequence of a massacre of Christians at Jeddah, and had been anchored off that port from October 1858 to March 1859. The ship's company was exceptionally good, and had enjoyed a remarkably high state of health, as may be gathered from the fact that, notwithstanding the intense heat and laborious work they had often to endure during the eighteen months since leaving England, the sick-list had not exceeded the ratio of 7 per cent. on strength, two-thirds of which were surgical cases; that the mortality rate was only 6 per 1,000, and the invaliding rate only 30 per 1,000, and in no case from serious climatic diseases. This satisfactory health-condition, unexampled, I believe, on that station, was due in the first instance to the excellent physique of the men, who had been carefully examined individually on entry, but mainly to the thorough good order and intelligent discipline that were maintained, and to the scrupulous attention that was given to cleanliness and ventilation. The lower parts of the ship were kept perfectly sweet, and pumped dry. The solid hatches had been removed from over the screw-passages, etc., and replaced by open gratings, through which the whole of the lowest part of the vessel was exposed to the fresh air, and the slightest impurity was readily perceived, and at once removed. The men, 500 in number, also enjoyed the inestimable privilege of abundance of distilled water, both for drinking and also for ablutions, when no rain-water could be had.

The climate of Jeddah during the winter half-year was found to be eminently salubrious, the sick-list was only 4 per cent. on strength, with not a single death. The *Chesapeake* left that anchorage on the 1st of March, on becoming the flag-ship of the China station, and arrived at Aden on the 6th of March. There were, on that day, only six persons on the sick-list, or less than 2 per cent. As the crew had had no leave on shore since August, permission was given them to go for a few hours, and nearly all availed themselves of the

opportunity of visiting the place. In their walk or ride to the town they were, of course, exposed to a hot sun, and the provisions, water, etc., they obtained there were said to be of inferior quality. There were, as might be expected, some cases of intemperance and irregularity, but they were comparatively very few. On the 11th of March a seaman was attacked with choleraic diarrhœa, which soon assumed the true cholera aspect. In the same night a sergeant of marine artillery was taken ill in the same way. Both had been on shore a few days before, and had suffered from bowel-complaint. The seaman rallied under treatment, and recovered satisfactorily. The sergeant was sent to hospital for better nursing, as the ship was in great discomfort, while coaling for departure. The hospital nurses being natives, it was thought advisable that two of his messmates should accompany him to help in nursing him. On the 14th of March the ship sailed, leaving him behind, moribund, the two men having returned on board. While on shore they had lived rather freely, and had, it appeared, taken their meals in the sick man's presence, and both had had slight diarrhœa. Next day one of them showed slight symptoms of cholera, which soon became intense, and carried him off in eighteen hours. The other soon recovered. Numerous cases of bowel-complaint occurred for some days after our going to sea, but none of a grave character.

Although Aden was said at the time to be healthy, it afterwards transpired that an epidemic six months before had carried off many natives, and a few sporadic cases had subsequently occurred, even to the date of our visit, but, being confined to natives, had excited little interest. Our men had therefore acquired the disease on shore from existing centres of morbid development, but, fortunately, there were but few instances of its gravest manifestation; whether from the smaller dose of the cholera poison imbibed, or the non-susceptibility to its action of the great majority of persons who were probably exposed to it. The excellent sanitary condition and regulation of the ship doubtless contributed to check its spread; and in this respect it differed from the outbreak I described as occurring at Memel, where the minor cases were far more numerous and severe, and where one case of true cholera type occurred in a man who had not been out of the ship, or exposed in any way to the endemic poison of the infected port. In both the *Archer* and the *Chesapeake* the visit to the shore led to the outbreak of the disease,—a warning as to the risk that is always run in communicating with cholera-stricken localities; but the

danger of its spread on board was minimised or nullified by the more or less complete hygienic conditions that existed to oppose and overcome it.

I shall now, with your leave, touch on the subject of malarious fever, and first as it occurs in one of its chief abodes, the West Coast of Africa. The *Firefly*, a steam sloop-of-war, with a crew of 80 white seamen and marines, and 30 Kroomen (native sailors), was employed on that station from October 1850 to July 1853, in the suppression of the slave-trade, and visited in that time nearly every part of the coast, from Portendik, on the confines of the Sahara desert in the north, to Cape Frio in the south, a range of forty degrees of latitude and thirty-five of longitude, embracing every variety of tropical climate. She also crossed to the Cape Verde and Bissagos islands and Fernando Po, and occasionally, as sanatoria, to Ascension and St. Helena, where alone the crew were allowed the privilege of leave on shore. Her head-quarters were Sierra Leone in the north, and St. Paul's de Loanda in the south; but, with the exception of the Niger, she was much in the rivers whence the slavers generally proceeded, and which were therefore narrowly watched. The ship was singularly healthy, the greatest possible care being taken by the commanding officers, experienced African cruisers, to mitigate the inevitable hazard of the climate. In this they succeeded admirably; for the sick-list, varying, of course, with the season and locality, was never excessive, ranging from 3 to 8 per cent. on strength, fully one-half of which were surgical cases. Only one death from disease occurred among the ship's company during the whole of that period, in an officer, to whose case I shall allude presently.

Febrile disease, which constituted 13 per cent. of the whole amount of illness, was chiefly contracted in the rivers. The most severe cases of remittent fever were derived from boating service, in which the crews were away from the ship for several days, exploring the creeks and corners of the rivers, where the slave-vessels were suspected to be lurking concealed by the luxuriant vegetation, and where the men, either landing or lying close to the banks, were specially exposed to malaria.

From December 1850 to July 1851, the *Firefly* was stationed at the mouth of the river Congo, anchored from one to three miles from the shore, and making occasional trips for twelve or fifteen miles up the river. Neither in her nor in the boats similarly stationed, well off the shore, did any malarious fever occur. But among those who formed the boat's crews that ascended the stream periodically, a con-

siderable proportion—one-third of the whole number—suffered from intermittent or remittent, the latter in a severe form, with much subsequent debility and tedious convalescence. They used to be absent for a fortnight, passing up the mighty river as far as Punta da Linha, a Portuguese factory some forty miles from the mouth. From the great depth and velocity of the current, they were obliged to anchor at night in close proximity to the shore, and were therefore much more exposed to febrific miasmata, generated in the narrower and more hemmed-in portions of the stream. They generally returned to the ship in good health, some days elapsing before they were stricken down. The period of incubation varied from seven to twenty days, ten or twelve days being the usual interval. In those who were natives of malarious districts, such as Romney Marsh in Kent, or who had suffered previously from ague, it was observed either that the intermittent type only of fever was evolved, or that the remissions were much more marked than in those, the great majority, who had had no such previous history. In most cases pyrexia ran high during the first week, remissions being imperfect or obscure. In some cases a crisis was observed from the fourth to the seventh day, marked oftener by an improvement in the aspect of the patient than by any decided abatement of the febrile symptoms. Simultaneous with this was frequently noticed a critical evacuation, either copious perspiration, or large excretion of urine or hæmorrhages from the bowels, nose, or mouth. All recovered satisfactorily, convalescence being much accelerated by a voyage in the open sea, well off the coast, or across to Ascension or St. Helena.

The only fatal case was that of an officer, who had not been out of the ship for some months, or exposed in boating duty to morbific emanations. He was a man, however, of intemperate habits, and consequently greatly impaired health. His symptoms were those of the other cases, but superadded was extreme irregularity of stomach, which rejected all ingesta. On the fourth day he vomited a considerable quantity of dark liquid, was deeply jaundiced, had violent delirium, followed by excessive prostration and death. This Congo experience is instructive as showing that the malarious poison caused three different and, as classed by nosologists, distinct types of African fever,—ague in those who had previously suffered from it, and seemed thereby protected from the severer forms;—remittent, with its long period of incubation, sharp fever, critical evacuations, and tedious convalescence; and in the last case, where intemperance and mental anxiety had lowered the vital powers

the cerebral and digestive functions were more seriously implicated, and symptoms of yellow-fever were developed. Twelve cases in all occurred among the boat's crews, but two-thirds of them escaped all malarious disease, though as much exposed as their comrades. Quinine, as was then the custom, was given to all daily as a prophylactic, but whether it had such effect is uncertain. The Kroomen did not suffer from malarious fever, but being natives of the humid, hot coast of Guinea, were much affected by epidemic catarrh when in the cooler, healthier parts of the coast, and one died of pneumonia. A case of sporadic cholera of Asiatic intensity occurred in a white off Liberia, but recovered. No disease of the kind existed in the vicinity.

I shall now conclude with a few remarks on malarious fever as I encountered it in Central America, and chiefly in the river San Juan de Nicaragua, which drains the lake of that name into the Caribbean Sea. The steam corvette *Archer*, of which former mention has been made, was, after the conclusion of the Russian War, ordered to the West Indies, and in July 1856 was stationed at Greytown, Central America, where she spent the hot rainy season, with occasional cruises on the coasts of New Granada and Honduras. The ship and crew were in much better condition than they had been in the Baltic during the first year of the war. The sick-list did not exceed five per cent. on strength, and more than half the cases were surgical. Febrile diseases were not uncommon, as might be expected in a crew of young men, few of whom had been out of Europe, on first encountering the excessive heat and humidity of the tropical summer. But they were mostly of ephemeral character, and readily amenable to treatment. At Greytown, which is at the mouth of the St. Juan river, there were fourteen cases of intermittent, of quotidian, and tertian type, and as many in which there was but one well-marked paroxysm, pyrexia being at the outset exceedingly high, but moderating after a few hours, and persisting, with some remissions, for some days, seldom exceeding a week, when the fever passed away and the patient got speedily well. In this respect it differed widely from the remittent fever I have described as found in the Congo and other African rivers, where, with febrile symptoms of apparently no greater intensity, the debility induced was generally very great, and the period of convalescence much more protracted. Intermittent and remittent fevers of the same kind are common among the residents of Nicaragua river; seldom fatal, except through the chronic hepatic, splenic, and anæmic disorders they engender.

As the ship was necessarily anchored near the shore she was exposed to the same malarious influences as the town. Unusual exertion, exposure to the sun and rain, and constipation appeared to be concurrent factors in determining the attacks, which affected not more than 16 per cent. of the ship's company during the nine weeks of her stay at Greytown in August, September, and October 1856.

One fatal case occurred which presented unmistakable indications of yellow-fever. We had left Greytown on the 19th of October, and after cruising along the coast had spent a week at Belize, in British Honduras, leaving that port on the 5th of November. On the 9th, at sea, a young officer was attacked with febrile symptoms. He had no distinct rigors, and pyrexia was not excessive. The case seemed in no respect to differ from some others on the sick-list at the time, of febricula from ordinary causes. Indeed, he seemed to be less ill than most of them, and his bowels being much relaxed, he had gone five or six times to the round-house on the upper deck during heavy rain. On the 11th he became worse, with typhus symptoms, and in the evening vomited a large quantity of black fluid. The skin was observed to be of a deep lemon colour, with dark-coloured petechiæ, and he died next day, on the fourth day of the disease. This officer had been on shore two nights at Belize, and had got wet after leaving a ball, overheated with dancing. He was of intemperate habits, and had indulged to excess on those occasions. In this particular the case was analogous to that of the young officer in the Congo, where chronic alcoholism seemed to be the chief agent in intensifying the fever and inducing the fatal symptoms. In this case at Belize there were the additional circumstances of his landing, and of having got wet in the tropical rains, both on shore and during his illness on board. On shore he may possibly have been exposed to the contagion of the yellow typhus, sporadic cases of which sometimes occur in West India ports. It is more probable that he had only the mild climatic fever of his shipmates, aggravated by special exposure, and by individual peculiarity of constitution. In the pathology and etiology of all diseases, more especially of the malarious and zymotic kind, individuality, hereditary proclivity, and personal susceptibility play a most important part in determining the attack, and can alone account for much that is otherwise inexplicable in the incidence of or exemption from those maladies.

Bilious discoloration of the skin, of such fatal augury in Atlantic fevers, is by no means so formidable a symptom in European epidemics. An interesting outbreak of jaundice,

reported not long ago to have occurred at Bremen in North Germany, the account of which by Dr. Lurman was translated by our colleague, Dr. Edwardes, seems to be well worthy of notice. It occurred in a large ship-building factory at Bremen, where 1,230 to 1,500 persons were employed, and affected 191 persons in six months, betwixt October 1883 and April 1884, most of the cases (137) occurring in December. It was confined to men, affected all classes, masters and *employés* alike, and seemed to be limited to this factory. There were a few cases in the town, but none in a neighbouring factory with 600 workmen; and no instance among the wives or children of the jaundiced men is recorded. The disease began with gastric and intestinal catarrh, anorexia, vomiting and constipation. After a week came yellowness of skin and diarrhœa. There was no fever, and the pulse was slow. No swelling was detected indicating enlargement of liver or gall-bladder; the fæces, at first pale, soon became normal in colour, notwithstanding persistent jaundice. No death occurred, and only one case of cholæmia with cerebral oppression. The etiology of this epidemic was most obscure. No malarial or dietetic cause could be assigned to it. It was then remembered that in that year, 1883, all the men in the factory had been revaccinated—in most cases, without effect. But of 1,340 thus vaccinated, 191 had jaundice, and marked differences were found in the ratio of attacks in different parts of the factory. In one portion, where 540 persons were employed, 141 became jaundiced; in another part, only one in 50. After this revaccination 500 persons were taken on at the works, no one of whom suffered from jaundice; and there were 87 persons who were vaccinated by other surgeons with other lymph, and no one suffered. This singular epidemic was thought to be an infectious disease, the virus to be strictly limited as to place and time, the incubation period to be two to eight months, and to be connected some way or other with the revaccination. The success or non-success of the operation seemed to be immaterial in relation to the infection with jaundice. In this connection Dr. Chauffard considers catarrhal icterus to be a general disease, probably caused by absorption of ptomaines found in the alimentary canal in consequence of abnormal fermentative powers. These ptomaines, acting on the liver, produce catarrhal inflammation and obstruction of the biliary ducts.

Our lamented colleague, the late Dr. Murchison, gave, in his classical work on liver disease, the history of several epidemics of jaundice, some with large, others without mortality; some, as at Bremen, confined to men; others, by pre-

ference, affecting children, and even pregnant women. Two of the most recent he narrates as occurring, one among the French troops at Pavia, in Northern Italy, in the war of 1859, when 70 out of 1,000 men were affected, and the disease was attributed to malaria, privation, and alcohol; and another at Rotherham, in Yorkshire, in 1863, where 150 persons suffered from it. And it was observed that such individuals had escaped the enteric fever that prevailed in the town in the previous year, which had been very fatal, and was traced to defective drainage.

I have trespassed too long on your patience, and will therefore refrain from dwelling on the remarkable extension of our knowledge of etiology through the inquiries of Drs. Ballard, Power, Cameron, Klein, and other workers, who have shown that milk has often played an important part in spreading among children diarrhœa and enteric fever, sore-throat, diphtheria, and scarlet-fever, with perhaps other maladies, sometimes through human, sometimes, apparently, through animal contagion. It is matter for congratulation that legal powers have been recently conferred on the local Sanitary Authorities to inquire into the condition of dairies, and to regulate the circumstances under which cows are kept; and it is to be hoped that this important matter will not be overlooked in future sanitary legislation for the Metropolis. The extension of microscopic investigation has revealed a world of minute living organisms, which are believed to be an essential part of some morbid processes. Further observation and experiment may prove this to be true. Such theories serve a useful purpose, as guiding biologists and pathologists to new methods of inquiry, resulting, it is to be hoped, in future success in the prevention and alleviation of disease. These researches will continue to afford food for interesting speculation and discussion in our Society.

TYPHO-MALARIAL FEVER.

By. J. EDWARD SQUIRE, M.D.Lond., M.R.C.P.

(*Read: December 8th*, 1886.)

I CONFESS that when I accepted the invitation of one of your Secretaries to appear before you, I felt that it was little short of presumption for one with only a few months' tropical experience to present a paper on typho-malarial fever to this Society, numbering among its members, as it does, men much of whose active life has been spent in tropical and sub-tropical countries, and some whose investigations and writings have added not a little to our knowledge of the subject under consideration.

Like many others who have seen enteric fever at home, and who have then had to deal with fever as it appears among troops set down in a tropical country, I found myself, when observing the fever which soon appeared in the camps round Suakim last year, first fully satisfied that I had to deal with enteric fever; and soon discovering my experience at fault by seeing cases, which I would fain have believed to be enteric, puzzling me by variations in symptoms or in the course they ran, and finally showing me at a necropsy an absence of the enteric lesions I had expected to find. Realising, then, that I had been witnessing and treating two distinct diseases, I commenced a search among the writings of observers who have recorded their opinions on fevers occurring under similar conditions. The result of this I now bring before you, rather than any large amount of my own original observation. It will be seen that I have ventured to differ in certain respects from some whose views are worthy of the greatest consideration, leaving it to those most qualified to judge whether I am justified in so doing.

And first it is necessary, before proceeding further, to understand what definition we intend to give to the term typho-malarial fever. This may be unnecessary when speaking of well-known and long-studied diseases; but we are here dealing with a condition little seen in this country, and on the nature of which there are several different opinions. If we follow the official nomenclature of the Royal College of Physicians we shall consider typho-malarial fever to be a

combination of malarial and enteric fevers. This agrees with the opinion recorded in the minutes of the meetings of the Section of Medicine at the International Medical Congress, held at Philadelphia ten years ago, namely, that typho-malarial fever is a compound fever resulting from the simultaneous action of two distinct poisons—that of enteric fever and malaria. With such weight of authority in favour of this view there would seem hardly room for any other opinion; yet it does not appear to me to be the right one.

A somewhat similar view to that mentioned above was upheld by Dr. E. G. Russell, in a paper read before this Society in 1881, in which he speaks of typho-malaria as an example of the parallelogram of forces, it being, as he considers, the resultant of two distinct poisons, the enteric and the malarial. Dr. Woodward, of America, to whom we are indebted for the name, would call it a hybrid between these two diseases. Sir Joseph Fayrer and M. Léon Colin, two distinguished members of this Society, consider it to be enteric fever modified or even *induced* by malaria; and this would represent the views of many of our Indian and Army Medical Officers. I am going to ask you to consider more fully another view of the nature of typho-malarial fever, and I shall ask you to give it preference over the others. It is no new idea; it is held by many of our Army Medical Officers, and is borne out by pathological examinations. This view is, that typho-malaria is not a result of the enteric fever poison, but a form of malarial fever. This was the conclusion come to by the Commission inquiring into the subject during the American war, who reported that they considered the camp fever to be bilious remittent, which had assumed the adynamic type found in enteric fever. I will, therefore, ask you to allow me to define typho-malarial fever as *an expression of the malarial poison (or a malarial fever) in which intestinal and adynamic symptoms are prominent, causing the illness to simulate enteric fever.* My paper will consist chiefly of a justification of this view.

There are some, like Virchow and Dr. Marston, who doubt the expediency of introducing the term typho-malaria into our nosology. If the term denotes nothing more than a modified form of enteric fever I think they would be right. We cannot sanction a fresh name for each modification of a specific disease. We have, certainly, "malignant" scarlet fever, and "hæmorrhagic" small-pox; if we require any name for enteric fever when modified by malaria, let us call it "malarial" enteric fever.

I take it that the College of Physicians, in saying that

typho-malarial fever is a combination of malarial and enteric fevers, intend to signify that the one is modified by the other; not that a distinct disease—a hybrid, in fact—is produced, as would be suggested by Dr. Russell's simile of the parallelogram of forces. It is surely time that the belief in hybrid diseases was past for ever. A specific poison produces a specific disease with certain pathological results; the symptoms may be modified by a variety of causes, acting either within or external to the patient. Two poisons may enter the system about the same time, and one may then delay or otherwise modify the manifestation of the other; but a new disease is not thereby produced by the conjoint action of these poisons. If the term typho-malaria expresses nothing to men's minds beyond a modification of enteric fever, or a hybrid disease, it is not worthy a place in our nomenclature. But if a poison absolutely distinct from that which produces enteric fever, and with a different pathological manifestation, may, under certain conditions, cause symptoms closely resembling enteric fever, and an illness often mistaken for this disease, then we have a morbid state of much interest and great importance, and one which has a claim to special recognition. To my mind this is exactly the case with regard to the subject of this paper, and for this reason I consider it worthy of a special designation. The term typho-malarial fever, having at length been included in our official nomenclature, will not, I hope, be expelled; but I also hope to see it transferred from its present position under enteric fever, and placed as a subdivision of malarial fever.

If we wish to trace the cause of a disease, it is to the pathological signs, rather than to symptoms, that we turn; and in order to decide as to whether the class of cases we call typho-malaria are due to the poison of enteric fever or to the effects of malaria, we must consult the record of necropsies.

I am aware that cases have been described as typho-malaria, in which the same lesions as are found in enteric fever have been present; but considering the similarity of symptoms, is it not quite possible that these ought to have been diagnosed as enteric fever? The symptoms may have been modified, but, as I have already said, that alone would not justify a different designation. I will attempt to show that, in this as in most other cases, pathological examinations are often the sole means of deciding the question of diagnosis.

In my own limited experience at Suakim, I came across cases of undoubted enteric fever, in some of which I was enabled to verify my diagnosis *post mortem*. Other cases I diagnosed as enteric fever, sometimes with hesitation, some-

times with my opinion unshaken till the illness had lasted some time, and in all cases without finding anything definite during life to make me doubt or alter the diagnosis. At last an opportunity occurred of testing such a case by a necropsy, with the result that I found a different pathological condition from that which I had expected. Instead of ulceration of Peyer's glands in the intestine, I found general congestion and ecchymosis of the intestinal mucous membrane, but with no breach of surface. This was clearly not a case of enteric fever, for the man had been ill three weeks. Yet the symptoms had led me to diagnose it as such.

Just such an incident happened in the Bengal Presidency, and is reported in the Appendix to the Army Medical Report for 1879. Two regiments, the 30th and 40th, were stationed in adjoining camps, and fever occurred simultaneously in both. The Medical Officers were able to visit both hospitals, and for a time it was considered that the fever was the same in the two regiments, and that it was enteric. *Post mortem* examination in the 30th Regiment showed that, in that regiment at all events, the diagnosis was correct. But soon an opportunity occurred of testing the point in the other camp, when it was found that the condition of the intestines in the fatal case of the 40th Regiment was quite different. In fact, the intestine in this instance was healthy. Evidently, then, this was not enteric fever; yet it was, until this necropsy, thought that the same fever was present in the two camps. In the American Civil War the absence of Peyerian ulceration of the intestine was noticed in many cases of the prevalent camp fever, for which the term typho-malarial fever had just been coined. In the Zulu War we find the same thing noticed; and among the observers quoted by Sir Joseph Fayrer in his *Climate and Fevers of India*, we find numerous instances of similar observations.

I am informed that exactly the same experience as I went through at Suakim occurred to the Medical Officers in the case of the 70th Regiment when stationed at Dinapore, Lower Bengal, in 1883, when side by side with true enteric fever, cases believed to be enteric during life, were found to want the pathological signs of this disease when the intestines were examined. Such, in fact, has been the experience which has led to the feeling of want for a special name.

That the differentiation of fevers in tropical countries is not yet complete, and that more than one condition is included in the fevers returned as enteric, has for years been clear to our authorities in the Army Medical Service. Some of these, classifying all the cases under this heading, have

held that the causation of enteric fever must be admitted to include climatic conditions, as well as unsanitary surroundings and specific germs. Others would go to the opposite extreme, and classing all the cases as remittents, would exclude enteric fever from their returns. Some affirm the existence of ulceration of Peyer's patches in all cases, whether climatic or specific. Others, while admitting the occurrence of ulcers in the bowels in the malarial fever, deny that this ulceration resembles that found in enteric fever. Before Scriven recognised enteric fever in India, all long-continued fevers in that country were considered to be malarial; and in the writings of Twyning and Annesley we find descriptions of diseases which they unhesitatingly call malarial, which we should probably have called enteric from their symptoms. But whereas in some of these ulcerations of Peyer's patches were found, others, almost identical in symptoms, showed no ulcers. Davies, in his work *On the Walcheren Fever of* 1809, describes cases illustrating the same point; and it is this occurrence of cases of enteric fever, side by side with malarial cases which simulate it, that renders the discrimination so difficult.

Let me now give a sketch of the disease as it shows itself during its course, and then speak of its pathological signs. Its onset, though insidious, like that of enteric fever, may seem more sudden from the occurrence of chills. Tonsillitis sometimes precedes the more marked illness. Vomiting is often an early and persistent symptom; the vomited matter being greenish, or sometimes, as I have seen it, quite clear, like a solution of sulphate of iron. This prominence or persistence of vomiting is one of the distinctive characteristics of the disease. Then, with increasing malaise, accompanied with pains in various parts of the body, and often attended with diarrhœa, the patient is admitted to hospital. Diarrhœa is not a constant symptom, just as it is not invariable in enteric fever; but, as it is usually the presence of diarrhœa with the other symptoms which suggests enteric, we must, according to our definition, consider the occurrence of diarrhœa as a frequent characteristic. The stools are sometimes greenish, often ochre-coloured, and closely resembling the evacuations of enteric fever. Sometimes, with diarrhœa as the usual condition, a solid motion may be passed in the middle of the illness. Occasionally, the congestion which causes the diarrhœa may extend throughout the whole length of the alimentary canal: we may then have mucous catarrh of the nose and fauces, or symptoms suggesting dysentery.

As the case proceeds, the appearance of the patient comes

still more to resemble that seen in enteric fever. The tongue may become dry and brown, and, with the teeth, is covered with sordes. The mental apathy of commencing adynamia appears, and delirium occurs at night. Later still, low, muttering delirium, with subsultus and other signs of the typhoid condition, give evidence of approaching death from exhaustion. Slight cases, showing convalesence in a few days, may have commenced with symptoms as severe as those ushering in an illness of months. In the more severe cases, the illness is usually long, from the prolonged convalescence. Patients are sometimes under treatment for three months before the absence of diarrhœa or of evening fever makes it possible to allow dismissal from hospital.

The temperature in these cases, though resembling that in enteric fever, often reaches a high point earlier in the disease, and the daily range is greater than in uncomplicated enteric. It must be remembered that the presence of malaria may increase the daily variation in temperature in undoubted specific enteric fever.

The abdomen is often distended and tender, and some observers have described rose-spots similar to those of enteric fever. I should hesitate to accept this until more cases have been recorded in which rose-spots occur without intestinal ulceration. I thought they were present in some of my cases at Suakim, but I am inclined to think that I was mistaken, and was misled by the presence of sudamina, which were so common under the cholera-belt, very generally worn. The occurrence of rose-spots in cerebro-spinal fever shows that enteric is not the only fever with such a rash; but I think we may take the presence of a rash to indicate the result of a specific poison.

It will be seen that we have here symptoms very closely resembling those of enteric fever, the points of difference being very slight—such as the vomiting, differences in the temperature chart, and perhaps the length of illness. As the two diseases—typho-malaria and enteric fever—may, and often do, occur side by side, it is evident that a diagnosis must be the result of weighing carefully all the various symptoms, and even then may be impossible. If there were no differences in the etiology and pathology of the two conditions it would be immaterial which name were given to the case; but if, as I believe, the etiology and pathology are distinct, it is important to study how to distinguish the two diseases. One practical difference will illustrate and give force to this remark, viz., that enteric fever is communicable from person to person, while typho-malarial fever cannot be so transmitted.

Pathology.—According to the view of typho-malaria that I hold, it is on the pathological signs that we must chiefly rely for the marked distinctions that separate it from enteric fever. Speaking generally, the difference is found in this—that whereas proliferation of the cells, with subsequent ulceration, in Peyer's patches and the solitary glands of the intestines is the invariable pathological sign or expression of the enteric fever poison, ulceration of Peyer's patches is very rarely, if ever, found after death from typho-malarial fever.

I do not say that *ulceration* is very rarely seen after typho-malarial fever, but that it is not such ulceration, either in site or character, as that of enteric fever. Ulceration is probably not uncommon in various manifestations of the malarial poison, but it does not select, and is not confined to Peyer's patches and the solitary follicles. These ulcers may be found in any part of the alimentary canal, and may be of any shape and almost any size. They rarely extend deeper than the submucous tissue, but may be deep enough to cause perforation.

The pathological signs most generally described are as follows:—

General injection of the mucous membrane of the alimentary canal, often most marked in the duodenum and upper jejunum, but affecting at different times the whole length of the tract. With this fulness of the vessels there may be ecchymoses under the mucous membrane at various parts in the intestine.

When ulcers are found they are generally irregular in shape, and do not select the closed glands of the intestine.

The mesenteric glands are often enlarged.

The spleen is generally enlarged; the liver frequently congested.

Among various complications that may be met with, hæmorrhage is not uncommon, and may come from the intestines or from the urinary organs. Epistaxis occurs frequently. Pulmonary congestion or pneumonia may come on during the course of the fever. Jaundice may be present from congestive closing of the gall-duct. Purpuric blotches or subcutaneous ecchymoses may be seen without scurvy, as in some of my cases in the Soudan. Woodward mentions the occurrence of scurvy in malarial fevers, and Dr. Harley speaks of it in connection with intestinal ulceration. Rheumatism, sometimes with effusion into joints, has been observed.

All these pathological conditions and complications will be recognised as occurring in other forms of malarial fevers. Dr. Maclean, in his article on "Malaria", in Reynolds'

System of Medicine, says: "Hyperæmia of the stomach and duodenum is one of the most common of the appearances found *post mortem* in intermittents." Typho-malarial fever may be met with wherever malaria is found: imperfect sanitation may determine the intestinal symptoms. Perhaps also the presence of enteric fever in the neighbourhood may have something to do with this. In fact, it has been suggested that typho-malarial fever should be described as the type taken by remittents in the presence of enteric fever. Over-fatigue and excitement, or any condition tending to produce bodily or nervous depression, may predispose to an attack, and this will account for its presence in camps, especially on active service. In such cases, the coexistence of enteric fever in the force, and the obstacles in the way of accurate and sustained observation, renders the discrimination more difficult.

It seems probable that many cases entered in the returns from our foreign stations as local fevers might correctly be called typho-malarial fever. Surgeon-Major Veale, in the Army Medical Report for 1879, considers that the terms Gibraltar fever, Malta fever, and Mediterranean fever are sometimes employed in describing cases of this disease. My own experience of this fever was gained at Suakim (of which place I have here a map); and, though the coral basis of the camp may seem opposed to its being considered a malarious site, the surroundings were such as to make malaria possible, and, in fact, experience proved it to have been present. In the Navy Medical Report for 1884, remittent fever is mentioned as occurring at Suakim; and Surgeon L. H. Kellett, M.B., who was there with the Marines, says as to this, that "sometimes it resembled enteric fever in everything but the eruption" (pp. 104-107).

I have endeavoured in this paper to give to the term typho-malarial fever a more defined and exact signification than it can be said to possess at present. In reading the accounts of cases published as typho-malaria, one is struck with the marked dissimilarity between them, both as regards symptoms and pathology. Some are apparently true enteric fever; others have no resemblance to that disease at all; some have intestinal symptoms only; in some the adynamic condition is marked, and still others are wanting in both these signs. In one or two, the length of illness and the indefinite nature of the symptoms have apparently led to a diagnosis of "typho-malaria" for want of a better designation. This uncertainty is, I am aware, more a matter of what cases shall be included under the name, than a doubt as to the appear-

ances found in the *post mortem* examinations. I would restrict the term to malarial fevers, which, from the prominence of intestinal or adynamic symptoms, closely simulate enteric fever; and I would exclude those cases which are found to present *post mortem* the pathological appearances of this latter disease.

APPENDIX.

The two following cases will serve to illustrate the important points in the temperature range of malarial fever and of typho-malarial fever. The first is a case of ordinary malarial fever recognised as such during life, and shows the wide diurnal range of the temperature. The second, as will be seen from the abstract of the case, comes under the definition given in the paper for typho-malarial fever, the diagnosis remaining uncertain till after death. It illustrates the irregularity and long continuance of the fever.

I.—MALARIAL FEVER.

Private J. S., *aged* 24, *40th Regiment, Choubattia.*

Date.	Day of illness.	Temperature.		Daily Range.
		A.M.	P.M.	Fahr.
June 28	3rd	98.	98.8	0.8
„ 29	4th	98.8	99.6	0.8
„ 30	5th	99.	100.	1.0
July 1	6th	99.	100.	1.0
„ 2	7th	98.	103.8	5.8
„ 3	8th	100.	103.	3.0
„ 4	9th	99.4	102.6	3 2
„ 5	10th	99.4	104.	4.6
„ 6	11th	99.4	104.	4.6
„ 7	12th	99.	103.	4.
„ 8	13th	98.6	103.	4.4
„ 9	14th	100.6	103.6	3.
„ 10	15th	100.	102.4	2.4
„ 11	16th	99.	101.6	2.6
„ 12	17th	102.4	103.	0.6
„ 13	18th	97.6	105.	7.4
„ 14	19th	98.6	102.8	4.2
„ 15	20th	97.6	101.	3.4
„ 16	21st	97.4	103.	5.4
„ 17	22nd	97.6	100.	2.4
„ 18	23rd	97.	100.	3.
„ 19	24th	98.6	100.4	1.8
„ 20	25th	99.	103.4	4.4
„ 21	26th	98.8	103.4	4.6
„ 22	27th	100.	102.4	2.4
„ 23	28th	98.	103.	5.0
„ 24	29th	99.	99.	0.
„ 25	30th	98.	102.	4.0

II.—Typho-malarial Fever.

Corporal W. J., *aged* 23, *40th Regiment, Chouhattia. Admitted for Ague on 19th March* 1880. *Disease changed to Enteric Fever 31st March* 1880. *On post mortem examination disease diagnosed as Remittent Fever. Died 10th June.*

Date.	Day of illness.	Temperature.		Daily Range.	Remarks.
		A.M.	P.M.	Fahr.	
March 26	8th	101.6	103.	1.4	
„ 27	9th	101.8	101.	0.8	
„ 28	10th	102.3	102.3	0.	
„ 29	11th	102.	102.	0.	
„ 30	12th	102.	103.4	1.4	
„ 31	13th	102.6	103.6	1.0	
April 1	14th	102.8	103.	0.2	Tongue brown and cracked. Stools pea-soupy and frequent.
„ 2	15th	101.8	102.4	0.6	
„ 3	16th	100.6	101.2	0.6	
„ 4	17th	101.	102.4	1.4	
„ 5	18th	101.	102.6	1.6	Hypostatic congestion of lungs. Bed sores.
„ 6	19th	101.	102.4	1.4	
„ 7	20th	101.4	103.	1.6	
„ 8	21st	101.	100.6	0.4	
„ 9	22nd	99.	103.	4.0	
„ 10	23rd	100.4	101.	0.6	
„ 11	24th	101.	102.6	1.6	
„ 12	25th	101.6	102.	0.4	
„ 13	26th	101.4	103.	1.6	
„ 14	27th	101.6	102.	0.4	
„ 15	28th	103.	104.	1.0	
„ 16	29th	101.6	104 2	2.6	
„ 17	30th	100.6	104.	3.4	
„ 18	31st	102.	103.	1.0	
„ 19	32nd	101.4	101.4	0.	
„ 20	33rd	101.	102.	1.0	
„ 21	34th	102.4	103.	0.6	Tongue dry. Delirious at night.
„ 22	35th	101.4	102.	0.6	
„ 23	36th	100.6	101.	0.4	Low muttering delirium.
„ 24	37th	99.6	103.4	3.8	Tremors of muscles. Motions passed in bed.
„ 25	38th	100.	102.6	2.6	Tongue dry and brown.
„ 26	39th	98.	101.4	3.4	Sordes on teeth and lips.
„ 27	40th	99.	103.	4.0	
„ 28	41st	98.4	102.4	4.0	
„ 29	42nd	98.4	100.6	2.2	
„ 30	43rd	99.	99.6	0.6	
May 1	44th	98.4	100.	1.6	
„ 2	45th	98.8	103.	4.2	
„ 3	46th	98.	102.	4.0	
„ 4	47th	97.	98.	1.0	
„ 5	48th	97.6	100.	2.4	
„ 6	49th	100.	101.4	1.4	
„ 7	50th	97.6	97.8	0.2	
„ 8	51st	100.	99.6	0.4	
„ 9	52nd	97.6	100.	2.4	
„ 10	53rd	99.4	100.4	1.0	

II.—TYPHO-MALARIAL FEVER *(continued)*.

Date.	Day of illness.	Temperature.		Daily Range.	Remarks.
		A.M.	P.M.	Fahr.	
May 11	54th	100.	99.	1.0	
„ 12	55th	100.6	101.	0.4	
„ 13	56th	101.	101.	0.	
„ 14	57th	101.	101.	0.	
„ 15	58th	101.	102.4	1.4	
„ 16	59th	101.	100.4	0.6	
„ 17	60th	100.2	100.	0.2	
„ 18	61st	101.	101.	0.	
„ 19	62nd	102.6	102.6	0.	
„ 20	63rd	100.6	100.6	0.	
„ 21	64th	100.6	100.6	0.	
„ 22	65th	101.	100.	1.0	
„ 23	66th	100.4	100.	0.4	
„ 24	67th	101.	101.	0.	
„ 25	68th	101.	101.4	0.4	
„ 26	69th	101.	101.6	0.6	
„ 27	70th	100.	99.6	0.4	
„ 28	71st	102.	101.6	0.4	
„ 29	72nd	100.	101.6	1.6	
„ 30	73rd	100.6	100.6	0.	
„ 31	74th	100.6	100.6	0.	
June 1	75th	100.6	100 8	0.2	
„ 2	76th	99.8	100.	0.2	
„ 3	77th	99.4	100.	0.6	
„ 4	78th	100.8	99.8	1.0	
„ 5	79th	100.	98.4	1.6	
„ 6	80th	101.	102.	1.0	
„ 7	81st	100.8	101.4	0.6	Sudden acute pain in right hypochondrium. Vomiting.
„ 8	82nd	99.4	98.	1.4	
„ 9	83rd	97.	97.6	0.6	Conjunctiva yellow. Vomiting continuous.
„ 10	84th	99.2	...	...	Died 2 p.m.

P.M.—Hypostatic congestion of the left lung; enlarged and nutmeggy liver; enlarged and softened spleen; congestion and mucous catarrh of stomach. *Large and small intestines healthy; no ulcers nor cicatrices.*

Both the above cases are taken, by permission of the Secretary of State for War, from the *Army Medical Report* for 1879, Appendix No. II.

PREVENTIVE INOCULATIONS.

By EDWARD FRANCIS WILLOUGHBY, M.B.

(*Read: Jan. 12th*, 1887.)

From time immemorial it has been well known that certain infectious diseases occur as a rule but once in a lifetime, one attack rendering the organism insusceptible of subsequent infection. This immunity, however, does not invariably follow, nor is it always absolute and permanent, for there are persons who never appear to acquire it in respect of particular diseases. It seems to become less complete, if not to be altogether lost, after a certain lapse of years, and very mild attacks certainly confer an imperfect and but short-lived exemption.

Why such diseases should so protect the individual against further infection, is a problem for which, until quite recently, no satisfactory or even rational explanation has been offered; but it is no doubt closely connected with the scarcely less remarkable fact that in all of them the morbid process tends to spontaneous extinction within a limited and, in some, a strictly definite time, never assuming a chronic character, as inflammatory diseases so often do.

Non-recurring diseases are all due, or presumably due, to the development of pathogenic organisms, but not all bacterial diseases are non-recurring, nor do they all terminate spontaneously. Thus, yellow fever, typhus, small-pox, measles, scarlatina, and several others, as well as the variolæ of other animals, and anthrax, swine-plague, rouget, etc., are non-recurrent; while cholera, diphtheria, and enteric fever, not unfrequently occur more than once in a lifetime; and erysipelas, septicæmia, and tuberculosis may run an indefinite course, ending in ultimate recovery or in death. In a discussion on diphtheria in this Society last year, I advanced a proposition, which I believe to have the character of a law—albeit an empirical one,—viz., that the more specialised or differentiated a disease, the greater the immunity conferred by a single attack.

The idea which underlies all protective inoculation, at least all hitherto practised, is the induction of the particular

disease in a form so mild or so modified as not to endanger life, yet sufficiently defined as to confer an immunity similar, if not equal, to that which follows an attack of the disease incurred in the ordinary way.

But so much confusion exists in the popular mind, and even, I fear, among such of our profession as have not turned their attention to the subject, that I think it will not be amiss, especially at the present time, when, as I believe, we are on the verge of a new and most important departure, to take a retrospective glance at what has been already done, and to indicate clearly the principles on which each procedure is based, as well as their mutual relations. Classification is essential to all scientific definition, and I will accordingly divide all protective inoculations that have been either practised or proposed into four or five classes.

The first postulate of a protective inoculation is that the virus to be inoculated shall be derived, directly or indirectly, from the disease it is intended to avert. Thus, sheep-pox will not protect against small-pox, nor *vice versâ*, any more than scarlatina, small-pox, and measles will antagonise one another. This may appear too obvious to call for so decided a statement, but it is not so when we remember that nearly all French, and a few English and German, physicians and veterinarians, still believe in the occasional spontaneous origin and the specific character of cow-pox; whereas I maintain, as I did in the paper I had the honour of reading here last year, that cow-pox in the form of vaccination protects against small-pox only because, unlike sheep-pox and some other variolæ, it is small-pox, though profoundly modified by transmission through the body of the cow. A clear apprehension of this postulate would obviate such blunders as the substitution of sheep-pox for so-called cow-pox as a material for vaccination, which has been attempted in Italy; the proposed vaccination of sheep, to protect them against their own pox; and the worse than useless inoculation of swine with Pasteur's cultivations of rouget, in the hope of protecting them against swine-plague, which is a disease more akin to enteric fever, and standing in a relation to rouget somewhat analogous with that of enteric fever to the scarlatina of man. Indeed, the description of cow-pox, in Mr. Fleming's recent letter to the *Lancet*, as a disease "distinct from and antagonistic to" small-pox is, if not self contradictory, opposed to all pathological experience and analogy. The second postulate is, that the disease which it is proposed to inoculate must be one of which an attack contracted in the ordinary way protects the individual against subsequent infection. Like the former pos-

tulate, this caution is not uncalled for, when we have seen Ferran practising wholesale inoculations with what purported to be cultivations of the cholera bacillus, and finding advocates in other countries as well as in his own; though, as our venerable friend, Surgeon-General Dr. Murray, in the *St. James's Gazette*, and I, in my rejoinder to Dr. Cameron in the *Nineteenth Century*, showed from Indian and American statistics, that cholera may attack the same individual several times; the sole reason why such repeated attacks are not more often observed in Europe and America, being the length of the intervals between successive epidemics. In like manner, Pasteur has proposed inoculations with cultivations of diphtheria, a disease, one attack of which, as Dr. Greswell showed us last year, renders the subject of it more susceptible to infection than he was before, and may, under certain unhygienic surroundings, recur even without a fresh infection.

Assuming that the diseases under consideration fulfil these conditions, we may endeavour to obtain protection by one of several methods which I shall, for the sake of conciseness, call the methods of inoculation, vaccination, attenuation, and, for want of a better term, neutralisation.

The first and second methods have as yet been practically employed only in the case of the pustular exanthemata or variolæ of man and animals, and the principles on which they are based are those that I laid down last year in my paper on Human and Animal Variolæ, which, with your permission, I will repeat:

1. One attack of variola, of the kind proper to any animal, protects the individual against infection by, or inoculation of, the same.

2. Inoculation of any animal with the virus of its own variola produces a milder form of the same disease, but affords a protection similar to that conferred by an attack contracted by ordinary infection. [This is the method of inoculation, properly so called.]

3. Any variola inoculated in an animal other than that whose proper variola it is, gives rise to a peculiarly modified form of the disease, attended by little constitutional disturbance; merely local eruption and no danger to life: such modified disease being no longer communicable to any other animal of the same or of different species, except by direct inoculation [cow-pox is such a modified form of small-pox]; and—

4. This modified disease affords a considerable degree of immunity against infection by any means whatever with the variola whence it was derived, either to the animals whose

variola was the original source of it, or to others capable of being infected in any way thereby. [This is vaccination, properly so called.]

I. Inoculation, or the induction of the identical disease in a milder form by means of virus taken from ordinary cases, has been practised in India, China, and the East generally, from the earliest times, as a protection against natural small-pox; the Chinese employing insufflation of the powdered scabs, and other nations inoculating the fluid of the pustules into the skin by means of needles or lancets. The practice was introduced into Europe by Lady Mary Wortley Montagu in 1723, though afterwards abandoned in favour of vaccination, and still later prohibited by law.

Ovination, or the inoculation of sheep with their own variola, was for some time largely practised in Germany, but has recently been declared illegal.

This method is open to the grave objection that, though when the operation is performed with skill and judgment death very rarely follows, and the individual acquires the highest degree of immunity attainable, the disease thus artificially induced has undergone no change, except as regards severity, and is liable to be communicated to others by the ordinary means of infection, resuming in such individuals its normal virulence. Thus, during the fifty years or so that inoculation was practised in England, the total mortality from small-pox was actually greater than when all alike were unprotected. The same result was found to follow the practice of ovination in Germany, and led in like manner to its ultimate prohibition; it being, too, evidently impossible to stamp out a disease by a procedure that presupposes its perpetuation. I must not, however, leave this subject without noticing an observation of our friend, Dr. Pringle, that when, as in some parts of India, the material used for inoculation is taken only from inoculated cases, the eruption that follows becomes at length almost as local as that of vaccination.

II.—The second class of preventive inoculations to which alone the name of vaccination may with any fitness be applied, are those in which the action of the virus is so modified by its having been passed through the organism of some animal of a very different species, that it produces only a trivial ailment, though giving a considerable degree of immunity against infection.

The only practical application of this method, as yet known, is the Jennerian operation. Nothing analogous has yet been suggested even for the prevention of sheep-pox, though it is by no means improbable that some animal could be found

which might play the same part in relation to the sheep that the cow has played, and the horse might play, to man. Indeed, I can only attribute to a want of a clear conception of the true nature of vaccination, as distinguished from mere inoculation, that no efforts have been made in this direction in Germany, where sheep-pox is far more prevalent and destructive than it is here.

Hitherto, as I said, the Jennerian operation has stood alone; but, in reviewing the evidence on the outbreak at Hendon, of what I have ventured to call Cameron's disease, it is impossible to resist the suggestion of an analogous procedure in the case of scarlatina. The inferences to be drawn from Dr. Klein's observations are: (1) that scarlatina, communicated to the cow by some means at present unascertained, assumes the less grave and merely, or nearly, local character of Cameron's disease; (2) that Cameron's disease can be communicated as such to man by direct inoculation; (3) that it is not an indifferent form of blood-poisoning or local irritation, but that it so far retains the character derived from its original source that, when deeply injected into the circulation, it produces, even in the cow, symptoms more closely resembling those of scarlatina; and that (4) when cultivated in milk it resumes its pristine virulence, inducing indubitable scarlatina in man, and in the cow a disease far graver than Cameron's, and attended by all the renal and other pathological changes characteristic of scarlatina. May not inoculation with Cameron's disease confer on man an immunity against scarlatina strictly analogous to that which cow-pox furnishes against small-pox? This, if proved, would be a vaccination in every sense of the word.

III.—The third class of protective inoculations are the so-called attenuations of M. Pasteur, as practised by him for anthrax, rouget, and some other diseases of domestic animals. They are based on the observation that the bacteria of these diseases, if cultivated for some time in artificial media, and under certain unfavourable conditions of temperature, etc., lose much of their virulence, and, injected into the body of an animal, induce a very mild form of the particular disease, which, however, suffices to render the individual insusceptible to infection. With a view of reducing so far as possible the danger to life inseparable from such procedures, M. Pasteur is in the habit of performing two operations at suitable intervals, using first a weaker attenuation, and, when this has had time to exert its influence on the organism, a stronger, which, without this precaution, would have led to dangerous, if not fatal, consequences; in short, as he alleges, attenuation

No. 1 renders the animal insusceptible to No. 2, and this to the original and unmitigated virus.

This attenuation of a virus by artificial cultivation, even when conducted under conditions inimical to its development, is by no means a constant phenomenon; most often bacteria, if they grow at all, retain their characters and virulence unimpaired; indeed, some critics maintain that his so-called attenuations are merely dilutions or mixtures of the specific microbes with others of a non-pathogenic nature. But, admitting, even for the sake of argument, that such attenuation is practicable, the whole procedure is uncertain and hazardous in the extreme. I do not deny—on the contrary, I believe—that he has proved by crucial experiments that he has rendered a large number of cattle and sheep absolutely insusceptible of infection or of inoculation by the virus of anthrax; but, having no means of accurately, or even approximately, standardising the strength of his attenuations, they as often fail to give the desired protection, and thus prove delusive, or they actually cause the death of the animals which it was intended to save from the possibilities of accidental infection. That this has occurred in numerous instances in Germany and in Hungary, even when the operation was performed by his own pupils, is notorious; and we know not how many such failures or accidents may not have been suppressed in France.

So, too, with regard to rouget. Dr. Salmon, in America, found that inoculations, with cultures prepared by Pasteur himself, proved fatal in a large proportion of cases, and led to a small outbreak, happily suppressed, among the swine kept for the purpose of experiments, with the danger of introducing a disease hitherto unknown in that country. Indeed, Dr. Salmon thought that the preparation purporting to be the weaker, was really the stronger of the two.

This uncertainty of the results is fatal to the general adoption of protective inoculations by attenuations among animals, and *à fortiori* to its ever being employed in the diseases of man. Besides, even if the risks at present incident to this method should be overcome, it is still open to the serious objection urged against inoculations with the original virus, that it keeps the disease alive and involves the constant possibility, not to say certainty, that it will be communicated to previously healthy herds and flocks by the ordinary means of infection,—actual contact, human intercourse, and infected premises or vehicles. In fact, nothing short of urgent necessity, as the preservation of hitherto healthy individuals in the midst of disease, can justify a resort to inoculations of the first or third class, whether in the case of man or beast.

IV.—The fourth and last method that I shall discuss, and that to make known which is the main reason for my taking up your time this evening, is the inoculation of sterilised culture-fluids, that is, of the products of the development of the bacilli without the bacilli themselves.

Some experiments pointing in this direction had long ago been made by Dr. Klein in this country, but the credit of first successfully applying it, as well as of establishing it on an intelligible and scientific basis, is undoubtedly due to Dr. Salmon, Chief of the Bureau of Animal Industry, in the Agricultural Department of the Government of the United States.

In the course of a large number of experiments instituted with the view of determining the identity or otherwise of swine-plague with rouget or rothlauf, the discovery of the specific bacillus of swine-plague, and the effects of inoculations, Dr. Salmon found that the bacillus, which was quite different from that of rouget, was cultivated with the greatest ease in nutritive gelatines, and that injections of such cultures were invariably fatal to pigeons,—animals extremely susceptible to the disease when artificially communicated. He further observed that when cultures were exposed for some hours to a temperature of 60° C. (140° F.) the bacteria were invariably killed, and sowings of fresh gelatines with the heated cultures were unfruitful. In fact, they were completely sterilised, though there was no reason to suppose that the chemical products, if any, of the development of the bacteria had, unless they were very volatile, been materially altered. He had already suggested that immunity acquired in the ordinary way in consequence of an attack of such disease, might be produced by the action of these hypothetical chemical products on the tissues, rendering them insusceptible to the action of the bacilli. If this were the true explanation, inoculations with the sterilised cultivations should do the same.

Accordingly, he performed the following experiment, among others. On Dec. 24, 1885, three pigeons, described as Nos. 8, 9, and 10, were inoculated with .8, 1.5, and .4 cc. respectively of the sterilised fluid; and another, No. 7, with 1.5 cc. of the pure fluid, into which no microbes had been introduced. No. 9, which had received the largest dose of sterilised fluid, was evidently sick next day, but recovered; the others did not show any signs of disturbance.

On January 11, 1886, No. 7, which had been inoculated with the simple fluid, and No. 9, which had had the largest dose of the probably insufficiently heated fluid, were treated to a

subcutaneous injection of .75 cc. of a liquid culture five days old, prepared from a potato culture of fifteen days previously, and, therefore, probably not so strong as one freshly prepared from the pig would have been. No. 7 died seven days later, and the bacteria were found abundantly in the tissues and organs which were more or less disorganised. No. 9, which had received the heated fluid before, was ill, but slowly recovered, with the loss of the use of its legs. When it was killed, fifteen days afterwards, it was in all other respects healthy and well nourished, but in its pectoral muscles he found two sequestra of dead tissue containing a number of lifeless bacteria, which failed to multiply in fresh culture-fluids. Evidently, the previous inoculation with sterilised fluid had enabled the tissues to resist the action of the bacteria, and had led to their death. The operation had been thus far successful, though, as it appeared, injudiciously executed.

A second series of experiments, performed with greater care, gave the most satisfactory results. The procedure was as follows :—

Pigeon No. 10, which had had on December 24 an injection of .4 cc. of sterilised culture-fluid, and three others, Nos. 11, 12, and 13, were all inoculated on January 21 with doses of 1.5 cc. of a culture of 14 days direct from the pig, but which had been maintained at a temperature of 58° to 60° C. for several hours, instead of for two only as on the former occasion. For three or four days they seemed dull and quiet, with ruffled feathers, but at the end of the week were quite well.

On January 29, Nos. 10, 11, and 12 received a second and similar dose, with little, if any, effect on their health. No. 13, having been fiercely attacked by its fellows and severely injured in the head, was let alone in a coop by itself.

On February 6 all four received another dose of the same kind and quantity as before, with still less disturbance of their general health.

Finally, on February 13, No. 8, which had had .8 cc. only of heated virus, fifty days previously; No. 10, which had had .4 cc. at the same time, and 1.5 cc. on the three subsequent occasions ; Nos. 11 and 12, which had had three, and No. 13, two such doses, with a control-bird ; No. 14, which had never been inoculated, were all subjected to injections of .75 cc. of strong virus,—a dose that had always proved fatal, except in the case of No. 9, already mentioned.

The control-bird died in twenty-four hours, and No. 8, which had had only .8 cc. of heated virus (evidently an insuf-

ficient dose) fifty days before, died in forty-eight hours. The others were even less affected than they had been by the protective inoculations, and remained perfectly well. Cultivations with blood from the hearts of the two that died were turbid with the bacillus of swine-plague by the very next day, though only the minimum quantity of blood practicable was employed for the purpose.

That micro-organisms in the course of their development either secrete, or in some way cause to be produced in the fluids in which they grow, some chemical substances, is a well-known fact, familiar to us in the phenomena of fermentations, putrefaction, nitrification, etc.; and that, in some cases, these products are inimical to the life of the organisms producing them, is shown by the arrest of the fermentation of saccharine solutions when the proportion of alcohol has reached about 14 per cent., which thus constitutes the maximum possible alcoholic strength of a natural wine.

That the spontaneous extinction of the morbid process in fevers might be brought about in a somewhat analogous manner, was implied in the zymotic hypothesis of Dr. W. Farr; but it did not in itself suffice to explain the phenomenon of subsequent immunity, since the chemical products, whatever they might be, must surely be eliminated from the living organism within a period infinitely shorter than that for which the immunity is observed to endure.

Dr. Salmon would substitute for the chemico-vital hypothesis of Farr, one more purely physiological. We know from a consideration of the development of neoplasms from the elements of the normal tissues, as of carcinomata from connective tissue, and epitheliomata from epithelium, that cells, when once they have acquired a special character, tend to perpetuate it by transmitting it to each successive generation to which they give rise in the process of proliferation. We have, then, but to assume that the effect of the products of the bacteria in the fluids of the body on the bioplasm of the cells, is such as to render them no longer capable of affording a suitable pabulum for the bacteria, which, consequently, die out, and that the cells, whose bioplasm is thus altered, tend to reproduce their like for a longer or shorter period. In course of time, however, this tendency may wear out, and the cells revert to their original character, when the individual becomes again susceptible of infection by the bacilli of the particular disease. In some persons, perhaps the healthiest, this reversion to the primitive character, and consequent renewed susceptibility, takes place in the course of a very few years; such was the late Rector of Rugby,

who died at the age of eighty-three, of his third attack of small-pox; and such I believe myself to be, who, having been twice successfully vaccinated, have twice since suffered from that disease.

The failure of very mild attacks of scarlatina, for instance, to protect against subsequent infection is illustrated by the case of Pigeon No. 8, which, having been inoculated with .8 cc. only of the heated culture fifty days before, died in forty-eight hours when inoculated with the strong virus.

The advantage presented by this method over inoculations with the actual virus, or with attenuations of the same, other than those analogous to vaccination, which last are no longer infectious by ordinary means, is, that the pathological phenomena induced by the products of the bacteria, whatever may be the nature or intensity of the constitutional disturbance, are not those of the specific disease, but of something actually antagonistic thereto.

This method appears also to be capable of wider application than the second, which requires the intervention of an animal of a different species capable of contracting the disease by inoculation, and when inoculated, of completely changing its character. Unless the cow should be found to constitute such an intermediary in the case of scarlatina, vaccinations will remain, as at present, available only for the prevention of small-pox, and perhaps, *mutatis mutandis*, of the variolæ of other animals.

Unfortunately, the lower animals are insusceptible in every way to all other non-recurring bacterial diseases of man, and man contracts such animal diseases as can be inoculated unaltered. In like manner, anthrax, swine-plague, etc., undergo no transformation or diminution of their fatal character when inoculated into other animals, as rabbits or birds, which are not naturally subject to them.

But, since many pathogenic organisms can be cultivated successfully in artificial media without change, and swine-plague has been shown to produce in such fluids the same chemical products that it does in the living body, all we have to do is to identify the bacillus of a disease, and to find a suitable medium for its cultivation. There is one disease which, highly infectious, with a mortality of fifty per cent., though conferring almost absolute immunity on the survivors, and endemic or epidemic over wide areas, at once suggests itself as a fit subject for research and experiment on these lines: I allude, of course, to yellow fever. If it be true that Dr. Domingo Freire has identified and succeeded in cultivating the bacillus, the road is already clear; while the ex-

treme fatality of the disease itself renders attempts at treatment by inoculation with "attenuated" cultivations so hazardous as to be quite unjustifiable,—though, if reports may be trusted, he has performed them with seemingly encouraging results. Salmon's method is not open to the same objections, and its application to yellow fever deserves, nay, demands, the most serious consideration.

Under circumstances of special danger, as those in which the medical and other attendants on the sick in hospitals, and even more in camps, are inevitably placed, it would be expedient to try the same means of protection against typhus; but I do not think that the other fevers are sufficiently infectious and dangerous to call for preventive measures of this nature.

With animals whose lives have only a money value, and the sacrifice, if such a result should follow, of a few individuals, would be outweighed by the preservation of the majority, ethical considerations have no place; and the authorities of the Brown Institution could not render a greater service to the agricultural interest than by instituting experiments with sterilised cultivations of every one of the non-recurring infectious diseases of our domestic animals.

Of course, I do not pretend to speak with the authority derived from a practical experience of bacteriological and pathological research; but journalists—who, as lookers-on, are able from their vantage-ground to take in a comprehensive view of what is being done in every quarter of the globe—may in the world of science, as of politics, do much to guide and instruct public opinion, more, indeed, than the actual workers, whose time and energies are wholly occupied in their special pursuits; and, if I shall have induced any such to turn their attention to this new field, I shall find my reward in the success which, I doubt not, will, sooner or later, crown their efforts.

THE HISTORY OF CHOLERA IN THE EAST.

By JOHN MACPHERSON, M.D.,
Inspector-General of Hospitals.

(*Read: February 9*, 1887.)

THE following remarks may be regarded as supplementary to a paper which the author had the honour of presenting to the Epidemiological Society some twenty years ago.

Some interesting additions have been recently made to the history of cholera in the East in early times, as set forth by Mr. Macnamara, and the present writer. For most of them we are indebted to the researches of Col. Yule. That officer's *Glossary of Anglo-Indian Terms* is full of general information; and the article under the head of "Mort de Chien" should be read by everyone interested in the subject of cholera. For a few notices we are indebted to Dr. Semmelinck, a retired Dutch military surgeon, who has recently written a book on the early history of cholera. The main object of that gentleman's work is to declare that the *Mordexin*, or *Mort de chien* of Goa, so graphically described in 1542, was not true Indian cholera at all. He has much satisfaction in showing that apparently true Indian cholera (notwithstanding the accounts of Bontius) was not comman in Java, or in Dutch vessels, in early times; nevertheless, although an unwilling witness, he adds a few new facts to our early notices. Neither the notices of Col. Yule, nor those of Dr. Semmelinck, carry us further back than others which have been already published, but yet they serve to fill up gaps in the chronology and in the geography of the disease. In the short list of notices that follows, it has been necessary to attach a query to two or three of them, as not being absolutely certain. Dr. Semmelinck has called attention to the use of the word "*Bort*" by the Dutch, to denote cholera generally, or, as he would say, bilious cholera; but he has to admit that in some cases the "Bort" was true cholera. Col. Yule, again, has called attention to a very early notice of many deaths in China from what is termed a *Pessima Maladia di Flusso*. This phrase bears a very close resemblance to the *Flux aigu*, a phrase very often employed by the French to denote *Mort de chien* and epidemics of cholera in the eighteenth century.

Chronological Table.

1517 (?). "Portuguese ship's crew arriving in China lost seventy men of Pessima Maladia di Flusso" (*Archivio Storia Italiano vita*, 33, Appendix, vol. iii, 1846. Gio di Empoli).

1599. *Mordexin in India* (Cartelli, 227).

1602. "In those islands off Arracan the action of *Mordechin* is to produce a sunken and slender pulse, with cold sweat, great inward fire, excessive thirst. the eyes sunken, great vomiting, with system *derribado* collapsed" (*Conti*, Dec. IV, liv. iv, cap. 10).

1610. Goa. "Il regne entre eux une autre maladie qui vient a l'improviste, ils la nomment *mordechin*" (*Pyrard de Laval*, ii, 19).

1670 (?). De Graaf, in a place in Bengal called Soepra, saw a case of disease named *maigola*, probably cholera (*Semmelinck*).

1679. Bort, or cholera, in Ceylon, mentioned by Dr. Pilat (*Semmelinck*).

1690. Rumphius and Valentin, though their works were published some time later, refer to *Mordexin* in Amboyna about this date.

1695-1712 (?). A. Blankert, Surgeon-in-chief, mentions Bort, or cholera, as a common disease in Java (*Semmelinck*).

1716. Island of Bourbon. "The extraordinary diseases of this country are the cholick, and what they call 'The Dog's Disease'" (Account of Island of Bourbon, in *La Roque's Voyage, etc.*, London, 1726, p. 155).

1736. Case of cholera on board the *Marquetti* (*Semmelinck*).

1753. Bort in Batavia. C. Kleynhoff (*Semmelinck*).

1781. "The plague is now broke out in Bengal, and rages with great violence. It has swept away already above 4,000 persons: 200 and upwards have been buried in the various Portuguese churches within a few days" (Hickey's *Bengal Gazette*, Calcutta, April 21).

1812. Sporadic case of *Mort de chien* (*Original Correspondence, etc.*, Edinburgh, 1846, p. 287).

1813. "*Mort de chien* is nothing more than the highest degree of cholera morbus" (James Johnson, *Influence of Tropics, etc.*, p. 405).

It may be further remarked that Col. Yule has found notices of *mordexin* in the various Vocabularies.

1712-21. Bluteau, *Vocabulario.*

1778. *Grammatica Indostan*, Roma.

1808. *R. Drummond.*

These additions to our knowledge of the history of cholera in the East are not very extensive, but they all go to confirm what we know from a hundred other sources—how a form of cholera was known in most parts of the East: a disease different from the ordinary ones of the countries, and always mentioned in addition to them.

It would be interesting to be able to pronounce positively that the disease described on board ship in China in 1517 was the same as that which we know to have been prevailing at the same time in Malabar. But it is not absolutely certain that it was so. After that time we know from Dr. Semmelinck that the countrymen of Bontius were well acquainted with a disease called Bort, bearing a general resemblance to the Mort de chien of India. Some of the more important notices are those which refer to the Bay of Bengal and the Indian seas.

We have a full and unmistakable account of an outbreak of the disease in the islands of Arracan in 1600 (in which I witnessed a similar one in 1841). In the year 1670 a Dutchman appears to have seen a case of the disease on the coast of Bengal. Both these notices came very conveniently, to supplement our knowledge of the occurrence of cholera in Bengal, where early notices of it have been very scanty, especially considering that the delta of the Ganges has long been considered one of the chief seats of endemic cholera.

Though we know that cholera was widely spread along the coast of India, the early specific mention of the disease in Ceylon, even under the name of Bort, in 1679, is satisfactory; and still more so is the account of the disease, occurring under the name of "Dog's Disease" (a unique rendering of Mort de chien), in the island of Bourbon in 1716. This is strongly confirmatory of the accuracy of the tradition mentioned by Inspector-General Burke, that cholera had visited the Isle of France in 1775, long before the outbreak of 1817 reached it. The extract given from Hickey's *Calcutta Gazette* affords a good supplement to what Warren Hastings had written about the disease coming up from Ganjam, causing much mortality in Calcutta, and passing on.

The Edinburgh letters give the same account as Sir E. Impey had done of the common occurrence of sporadic cholera in Calcutta; while Dr. James Johnson's notice in 1813 shows that he believed there was an ordinary cholera morbus and an intensified one, the latter of which was the Mort de chien.

Some sceptical critics of the facts, in the *Annals of Cholera*, have supposed that, whenever the author found traces of the

occurrence of cholera, he imagined and meant to say that on each such occasion cholera had prevailed epidemically. On the contrary (although, when the data are so imperfect, it is most difficult to distinguish sporadic cholera from the same disease prevailing epidemically, either locally or generally), he marked with an asterisk those years in his chronological table of cholera, in which it seemed fair to suppose that the disease had been widely epidemic. He has no doubt that epidemics were tolerably frequent; but evidence is often scanty, and not of so full and convincing character as the description of the great epidemic at Goa in 1543; which, however, has failed to satisfy the requirements of Dr. Semmelinck's critical spirit.

It may be said that there is sufficient evidence of the disease having been about a dozen times extensively epidemic before 1817.

The disease, then as now, was at times sporadic, at other times epidemic. The disease might, as already said, be widely epidemic, or only locally so. It might, or it might not, travel far from home; according, we may perhaps say, as circumstances were favourable or unfavourable to its propagation. That the disease was often widely epidemic, is plain from the fact that the natives of India often applied to its outbreaks the phrases of *Murree* and *Ouba*, both of which are applied to pestilences.

In conclusion, with reference to Dr. Semmelinck and a few others, who will not see cholera where all its symptoms are not enumerated, it is to be borne in mind, that the great majority of early notices of the disease are by travellers, or by captains of ships. Of some seventy-five notices of the disease before 1800, only about one-third are by medical men. Under these circumstances, it is, to my mind, very remarkable that the leading symptoms are so well characterised, as at once to carry conviction as to the nature of the disease described to anyone who has had a fair amount of practical experience of Indian cholera.

RECORD OF CASES TREATED IN HOSPITAL DURING THE SMALL-POX EPIDEMIC IN WEST HAM IN 1884 AND 1885.

By JOHN MOIR, L.R.C.P.Ed.

(*Read Feb. 9th*, 1887.)

ABSTRACT.

In bringing before the notice of the Society a statistical account of the cases of small-pox treated by me in the West Ham Hospitals in 1884-1885, I may say that I had hoped, through the co-operation of the other medical practitioners in West Ham, to have been able to have given a practically complete account of the epidemic; and, with that object, I issued seventy-four printed circulars asking for information as to cases not treated in hospital, but as I only received three replies which were available for statistical purposes, I have been obliged to confine myself to the hospital records.

These, however, it will be found are in accord with the statistics of other small-pox hospitals, notably those published by Dr. Gayton. For this reason they have an undoubted value. I have followed as far as possible the lines laid down by Dr. Gayton in his statistical table, so that my cases may be an additional record to the large number, over 10,000, adduced by him.

Of 1,211 cases treated in the West Ham Board of Guardians' Hospital at Plaistow, twenty-six of the cases were said to have been revaccinated, and one of them died, but the death was owing to pneumonia, from which he was suffering on admission to the hospital, and not to small-pox, and only seven of the twenty-six bore marks of revaccination. Of those not revaccinated but with three or more good primary vaccination marks about 4 per cent. died; with from one to two marks 15 per cent., in the case of the imperfectly vaccinated 29 per cent., of those said to be vaccinated but without evidence 44.6 per cent., and of the unvaccinated 56.22 per cent. died; 132 unvaccinated males were admitted with 76 deaths, and 101 unvaccinated females with 55 deaths, a total of 233 unvaccinated cases with 131 deaths; the total number of cases being 1,211, with 237 deaths=19.6 per cent.

I have no hesitation in saying that the protection against

death by small-pox afforded by vaccination is in exact ratio with the efficiency of the operation, and that revaccination confers practical immunity from confluent small-pox, or from death by small-pox.

The statistics of 303 cases of small-pox treated by me in the Plaistow Cottage Hospital of the West Ham Local Board of Health in 1885, agree with those obtained in the Guardians' Hospital during the two years 1884-85, and give altogether a total of 1,511 cases, with a mortality of 18.4 per cent.

Of the 1,511 cases, fifty-seven were hæmorrhagic; five were complicated with phthisis; three with peritonitis; seven with pneumonia; one with diphtheria; two with convulsions; one with ophthalmia and loss of sight of both eyes (unvaccinated), and five with loss of sight of one eye. Two cases occurred of children born alive in the hospital, vaccinated, and escaping the disease, being discharged well on the mothers leaving the hospital; and one where the child was discharged from the hospital unvaccinated, developed small-pox some four or five weeks later, and then died of this disease. Several women were confined of dead children, and recovered. Three, however, suffering from hæmorrhagic small-pox, died. Two cases of hæmorrhagic small-pox recovered, but one of them died of cancer of the breast nine months afterwards. The death-rate was increased by the compulsory overcrowding of the hospital in December 1884 and January 1885, and fell again when the overfull state of the hospital was put a stop to by the opening of the Local Board Hospital in March 1885.

Another question has arisen in connection with small-pox hospital treatment, that of the occurrence of an exceptional number of cases in the immediate proximity of the hospitals. A record of the cases occurring in the neighbourhood of the Plaistow hospitals shows that Mr. Power's conclusions as to small-pox hospital influence are correct; and in the case of the Boleyn Roman Catholic Reformatory, which was invaded by small-pox on two different and widely separated occasions, it appears most probable that the outbreak arose from aerial infection from the West Ham Guardians' Hospital, which was situated near to it. A site which was at one time thought by the Local Board to be suitable for a small-pox hospital, was, therefore, given up on account of its proximity to one of the West Ham Board Schools. The new Town Council of the Borough of West Ham have at present under their consideration the advisability of building a permanent hospital for infectious diseases, and have instructed the Sanitary Committee to look out for suitable sites, bearing in mind

the Report of Mr. Power presented to the West Ham Board of Guardians last year. (This Report is appended.)

The condition of the West Ham community as to vaccination has been recently ascertained by a house to house inquiry under the superintendence of Dr. Kennedy, from whose report I extract the following particulars:—

"The Visitation extended over a time dating from the 30th day of March until the 14th day of May 1885, and during that time 25,267 houses were visited, and the statistics of 142,220 persons were obtained.

"Of the above persons, 140,186 were found to have been vaccinated, or 98.6 per cent. of the whole. 25,102, or 17.7 per cent., were found to have been revaccinated. 2,034, or 1.4 per cent., were found to be unvaccinated. Of the last, 817 were over the age at which the Vaccination Act requires children to be vaccinated.

"Out of 140,186 who had been vaccinated, 6,232, or 4.4 per cent., had had small-pox at some time during their lifetime, and out of 25,102 who had been revaccinated, only 25, or .09 per cent., had had small-pox at some time during their lifetime, showing that proper revaccination is an almost certain preventative against small-pox. On the other hand, out of 817 unvaccinated persons, no less than 427, or 52.2 per cent., had had small-pox at some time of their lifetime.

"Information was only refused in 175 cases."

APPENDIX.

Copy of Mr. Power's Report on Small-Pox at West Ham.*

As directed by your Minute of 10th December, I have inspected the hospital at Plaistow, which, in 1884, was hired by the Asylums Board of the Poplar District Board of Works for use as a Metropolitan small-pox hospital. And I have inquired respecting the behaviour of small-pox in the West Ham Urban District during the epidemic of 1884-5, with special reference to the concern that this and the other two Plaistow Small-pox Hospitals may have had in the small-pox incidences witnessed.

For a general description of the Poplar Hospital, I refer to a letter dated 20th January 1881, from the clerk to the Poplar District Board of Works, addressed to the Asylums

* Obtained from the Board of Guardians of West Ham.

Board, a copy of which letter is appended to this Report. Attached to this letter you will find a block plan of the site, and I annex a map of West Ham (six inches to the mile) showing the relations of this and two other Plaistow Small-pox Hospitals to the Urban Sanitary District.

You will note that the Poplar Hospital, the West Ham Guardians' Hospital, and the Cottage Small-pox hospital of the West Ham Urban Sanitary Authority are situated near together, on the extreme eastern border of West Ham parish (the Urban Sanitary District), about 100 yards or so north and north-west of the Barking high-road. The Poplar Hospital is bounded on three sides by roads, two of which have become built upon on their sides furthest from the hospital. South of the hospital, and within 30 feet of it, is a cottage in a triangular garden, which is on one of its sides coterminous with the hospital bounndary. The site of the hospital is only about ¾ of an acre, and barely suffices for the hospital buildings crowded upon it. Thus the external wall of the northern pavilion is only 20 feet from southern roads, while the western extremities of both pavilions are not 10 feet from Short Street. North-east of the hospital certain "receiving-rooms" are less than 20 feet from Western road; on the south the external wall of the administrative block is on the extreme boundary of the hospital site.

The Poplar Hospital, though consisting of two separate pavilions, each of two storeys, has only single administrative offices, and so far has never been made to accommodate at one and the same time patients suffering from different infectious diseases. Indeed, it has, since its erection in 1879, only been utilised on three separate occasions. As the Poplar District Hospital, it received, between May 1881 and May 1882, 165 small-pox cases; and between October 1882 and May 1883, 22 cases of scarlet-fever. As a Metropolitan Small-pox Hospital, under the Asylums Board, it had (according to Metropolitan Asylums Board Returns) under treatment between 31st May 1884 and May 1885, 645 small-pox cases.

The small-pox epidemic of 1884-5 has fallen on the West Ham Urban Sanitary District with very peculiar severity. In a population estimated in 1885 at 160,000, it caused in the two years above 650 deaths, a rate annually of two in every thousand of the inhabitants. Small-pox in West Ham, as in the Metropolis, began to be abundant about March 1884, and during April, May, and early June attained epidemic prevalence. Then for a while, as, indeed, is usual in London during the summer season, the disease abated

somewhat in this district; but with autumn it acquired renewed activity, and during winter and ensuing spring attained epidemicity not often witnessed at the present day in this country. In six consecutive months, November 1884 to end of May 1885, small-pox caused above 400 deaths in West Ham, a rate of mortality equal to 5 per 1,000 annually of the population.

The small-pox epidemic in question has not equally affected all parts of the West Ham Urban Sanitary District. It has fallen with exceptional severity on Plaistow, especially on that part of it in the neighbourhood of the three small-pox hospitals. On the annexed map, the circle of ¾ mile radius,* with its centre at the small-pox hospitals, comprises 5,051 houses in West Ham, with a population (estimated at six persons per house) of 30,306. The remainder of the Urban Sanitary District includes 21,959 houses, with (at a like estimate) 131,754 inhabitants. In the former area, which may be termed the hospital area, the small-pox death-rate during 1884-5 amounted to 6.4, and, in the latter, to not more than 3.4 per 1,000 of the population. There were not, so far as I can ascertain, any differences in the populations of the two areas of a sort to account for the exceptional small-pox mortality witnessed in the neighbourhood of the hospitals.

In reference to the concern of the Metropolitan Asylums Board (Poplar) Hospital and of other Plaistow Small-pox Hospitals in this excessive small-pox mortality in their neighbourhood, I append a table, showing, fortnight by fortnight, the relations in time between use of the hospitals in question and incidences of small-pox mortality on the hospital area, and on the remainder of the West Ham Urban Sanitary District.

From this table it would appear that:—

(1.) In the six months—December 1883, to 21st June 1884—corresponding to the rise and culmination of the first part of the epidemic in the metropolis—West Ham became notably affected by small-pox, especially, and earliest in that portion of it situated within ¾ of a mile of the Plaistow Small-pox Hospitals. During the period this hospital area suffered a rate of small-pox mortality ten times greater than that of the remainder of the district. The excessive incidence on the hospital area was in no way attributable to the

* A circle of ¾ mile radius is here used for the reason that it includes the whole of the built-upon area of Plaistow, and that while excluding the populous parts of Stratford, West Ham, and Canning Town, it leaves between these places and Plaistow a zone that is comparatively sparsely populated.

Metropolitan Asylums Board (Poplar) Hospital or to the West Ham Urban Sanitary Authority's Cottage Small-pox Hospital.

The former did not come into use until 31st May, and cannot, therefore, have operated to produce death from small-pox until quite the end of the period in question, and the latter had not yet been established. In so far, then, as the small-pox mortality of West Ham in this period was attributable to hospital-treated cases of the disease, it was due to the Guardians' Small-pox Hospital that had come into use before Christmas, 1883, and which, until March 1884, had mainly served for reception of small-pox cases from parts of West Ham Union other than the Urban Sanitary District of the same name which actually contained the hospital. From December 1883, to 21st June 1884, the Guardians' Small-pox Hospital at Plaistow admitted above 100 small-pox cases.

(2.) In the two-and-a-half months, 22nd June to 30th Aug. 1884, a period corresponding to the normal seasonal decline of epidemic small-pox in the metropolis, West Ham suffered about the same amount of fatal small-pox as in the preceding six months. But in this period the small-pox mortality in the hospital area was smaller, whereas in the remainder of West Ham Urban Sanitary District it was greater than in the antecedent period. Nevertheless, the actual rate of small-pox mortality in the hospital area in the period was nearly twice greater than in the remainder of the district. In this period the Metropolitan Asylum Board (Poplar) Hospital and the Guardians' Small-pox Hospital together received (in nearly equal numbers) about three times as many small-pox patients as were in the whole of the previous six months admitted to the Guardians' hospital. Temporary abatement, therefore, at this season of small-pox mortality in the neighbourhood of these hospitals was in no way connected with disuse of them for small-pox; the circumstance, however, is of especial interest, as being altogether confirmatory of the experience of Fulham, where, in the corresponding period of 1884, small-pox declined, and, for a while, even died out in the hospital area, notwithstanding that admissions to the Fulham Hospital in the period had increased threefold.

(3.) In the succeeding six months, end of August 1884 to end of February 1885, a period corresponding to that of renewed autumn and winter activity of small-pox in the metropolis, small-pox fastened with peculiar severity on West Ham, and especially on that portion of it situated within three-quarters of a mile of the small-pox hospitals. In this period the rate of small-pox mortality in this Urban

Sanitary District was nearly four-and-half times greater than in the whole of the preceding eight-and-a-half months. Meanwhile the rate in the hospital area increased again to more than three times that of the rest of the district. Throughout this six months the Metropolitan Asylums Board (Poplar) Hospital and the Guardians' Hospital were in active use for small-pox, each receiving 400 or more cases. The Cottage Small-pox Hospital of the West Ham Urban Sanitary Authority was not yet in use, but toward the end of the period it was prepared for small-pox and began to receive cases early in March.

(4.) In the next six months, March to September 1885, a period corresponding to that of spring exacerbation, followed by rapid decline and practical cessation of small-pox in the metropolis generally, the epidemic attained its *acmé* in West Ham, and then quickly declined and died out. In this period the rate of small-pox mortality for the Urban Sanitary District was $1\frac{3}{4}$ times that of the preceding six months, and nearly $1\frac{1}{2}$ times that of the whole of the previous epidemic. Meanwhile the small-pox death-rate in the hospital area, and that in the remainder of the district, became more closely alike than at any previous period of the epidemic, the rate in the hospital area was only one-tenth greater than in the remainder of the Urban Sanitary District. This approximation of the rates of the two divisions of the Urban Sanitary District was due, not so much to diminution of small-pox in the hospital area as to a very serious growth of the disease beyond, viz., in and about the Canning Town and Victoria Dock district, situated one or two miles south-west of the Plaistow Hospitals. At the very beginning of this period the Cottage Small-pox Hospital of the Urban Sanitary Authority began to receive patients, and thus for a while there were three small-pox hospitals at one and the same time in use at Plaistow. But this state of affairs did not continue beyond the middle of May; the operations of the Metropolitan Asylums Board (Poplar) Hospital were at no time so extensive as they had previously been; only about 80 cases are recorded as received there, and at the end of the month the Asylums Board lease of the building lapsed, and the hospital ceased to receive small-pox. Throughout the period (March to September) the Cottage Small-pox Hospital of the Urban Sanitary Authority and the Guardians' Small-pox Hospital continued to receive cases, and the latter remained in limited use for small-pox after closure of the former in November.

Extracted from the Sixteenth Report of the Medical Officer of the Local Government Board.

Small-pox Deaths of the Metropolis.	Small-pox Cases admitted to Plaistow Small-pox Hospitals.			Fortnight ending.	Small-pox Deaths in 1884-85 of Persons resident at Date of their Attack in West Ham Urban Sanitary District.					
					In Area within three-quarters of a mile of the Plaistow Small-pox Hospital.			In the remainder of the West Ham Urban Sanitary District.		
	Metropolitan Asylums Board (Poplar) Hospital at Plaistow.	West Ham Guardians' Hospital at Plaistow.	West Ham Urban Sanitary Authority's Cottage Hospital at Plaistow.		Number of Deaths.	Deaths per cent. of Houses.		Number of Deaths.	Deaths per cent. of Houses.	
				1884.						
8	—	6	—	5th Jan. ..	1	.019		—	—	
7	—	2	—	19th „ ..	—	—		—	—	
3	—	6	—	2nd Feb. ..	—	—		—	—	
6	—	2	—	16th „ ..	—	—		—	—	
12	—	5	—	1st March .	1	.019		—	—	
12	—	8 (101)	—	15th „ ..	1	.019		1	.004	
14	—	13	—	29th „ ..	1	.019	.310	—	—	.029
24	—	12	—	12th April..	3	.059		1	.004	
24	—	17	—	26th „ ..	2	.039		1	.004	
30	—	10	—	10th May ..	3	.059		1	.004	
66	—	20	—	24th „ ..	2	.039		1	.004	
103	32	11	—	7th June ..	1	.019		2	.009	
89	87	11	—	21st „ ..	1	.019		—	—	
90	17 (166)	14	—	5th July ..	—	—		1	.004	
61	15	16 (132)	—	19th „ ..	1	.019		1	.004	
53	3	36	—	2nd Aug...	—	—	.156	4	.018	.080
31	12	44	—	16th „ ..	1	.019		8	.036	
35	5	24	—	30th „ ..	6	.118		4	.018	
25	10	14	—	13th Sept. ..	1	.019		4	.018	
24	35	29	—	27th „ ..	4	.079		4	.018	
27	38	21	—	11th Oct. ..	—	—		7	.032	
48	54	36	—	25th „ ..	3	.059		5	.022	
66	20	45	—	8th Nov. ..	3	.059		3	.013	
95	31	15	—	22nd „ ..	12	.237		5	.022	
101	35 (397)	63 (461)	—	6th Dec. ..	9	.178		12	.054	
113	24	51	—	20th „ ..	12	.237	1.836	20	.091	.569
				1885.						
90	30	53	—	3rd Jan. ..	8	.158		11	.050	
112	43	43	—	17th „ ..	11	.217		10	.045	
117	32	30	—	31st „ ..	7	.138		17	.077	
116	40	37	—	14th Feb. ..	18	.356		16	.073	
81	20	32	—	28th „ ..	5	.099		12	.054	
62	17	33	27	14th March .	6	.118		27	.123	
47	14	42	21	28th „ ..	5	.099		30	.136	
79	6	47	17	11th April..	13	.257		29	.132	
100	19	41	22	25th „ ..	11	.217		28	.127	
114	6	43	23	9th May ..	8	.158		27	.123	
125	—	28	29	23rd „ ..	5	.099		36	.164	
117	—	42	38	6th June ..	7	.138		34	.154	
76	— (82)	44	31 (299)	20th „ ..	4	.079	1.519	17	.077	1.383
67	—	26 (493)	17	4th July ..	7	.138		21	.095	
38	—	45	26	18th „ ..	3	.059		20	.091	
33	—	20	12	1st Aug. ..	4	.079		12	.054	
29	—	15	11	15th „ ..	2	.039		14	.063	
14	—	9	8	29th „ ..	2	.039		3	.013	
15	—	12	8	12th Sept. ..	—	—		6	.027	
19	—	10	4	26th „ ..	—	—		1	.004	
10	—	2	4	10th Oct. ..	—	—		—	—	
—	—	2	1	24th „ ..	—	—		—	—	
	645	1187	299		194	3.840		456	2.076	

FURTHER OBSERVATIONS ON THE FILARIA SANGUINIS HOMINIS IN SOUTH FORMOSA.

By J. WICKHAM MYERS, M.D.

(*Read by* Dr. Stephen Mackenzie, *March 9th*, 1887.)

During the four years that have elapsed since the publication of my last paper* on this subject, I have from time to time been engaged in making the further observations which I now venture to record, and simultaneously have lost no opportunity of re-investigating those already communicated, aided in the latter by the invaluable criticisms my former paper elicited. I can only re-affirm my conviction as to the absence of the Filaria Sanguinis Hominis from this island, and that this is, in all probability, attributable to the non-existence of a suitable intermediary host for the embryo.

On several occasions supplies of mosquito larvæ were got over from Amoy, some of which came to maturity, thus enabling me, in a few instances, to watch the embryo filaria during one or two days of intra-mosquito existence, contrasting the inhospitality shown to the parasite by the native insects under observation at the same time. Incomplete as the experiments necessarily were, by reason of the limited life vouchsafed the mainland mosquito over here, still for two or three days they afforded me opportunity of again convincing myself as to the accuracy of Dr. Manson's observations and descriptions. Under ordinary circumstances I should have thought this reiteration unnecessary, but from a quotation given in Dr. Manson's paper read before the Linnean Society,† Professor von Leuckart is reported by Herr Scheube of Leipsic, when lecturing on the blood filaria, to have written as follows:—"Myers wollte dessen versuche auf Formosa, wo die filaria-krankheit selbst nicht autochtron vorkommt nachmachen, kam aber zu dem resultate, dass die filaria-embryoren von den muskitos volständig verdaut wurden."

From this it would seem that the distinguished writer has

* *Cust. Med. Reports*, No. 21 (1881), pp. 1-25; and *Trans. Epidem. Soc.*, vol. i (1881-82), p. 126.

† *Trans. Lin. Soc.*, vol. ii, pt. x, p. 363.

misunderstood my remarks, and been led to believe the very opposite of that I am bound to assert was my object to convey. Thus, though the absence of the Filaria Sanguinis Hominis from South Formosa was recorded, still I ventured to suggest, with some degree of probability, that the species of mosquito found here differed from that found on the mainland, inasmuch as they "digested instead of nurturing" the filaria embryo. In support of this view the various attempts made to filariate monkeys were described, and their failure shown to depend on my inability to obtain a species of mosquito in the island capable of performing the part of the intermediary host, or preserve those imported from Amoy long enough to effect perfect maturity of the filaria embryo.

It may be further mentioned that I happened to be in Amoy when Dr. Manson was collecting the specimens of mosquitoes he afterwards forwarded to the late Dr. Cobbold, and which that eminent helminthologist used in London to demonstrate the changes described by Dr. Manson as occurring in the filaria, while in the mosquito; and I most cheerfully admit that on more than one occasion the daily metamorphoses were distinctly seen by me, and further, that I repeated the observation up to the third day in Formosa, with mosquitoes obtained from Amoy, and arrived at precisely similar conclusions. Further extensive and careful investigation here has failed to discover a mosquito like the Amoy filaria-nurturing variety, either in species or capacity for nursing the blood embryo. My searches have included many and distant regions of South Formosa, and to the kindly aid of the Rev. David Smith, late of the English Presbyterian Mission, a no less constant than indefatigable traveller to the interior, I am indebted for specimens collected in various localities. This gentleman succeeded in obtaining one variety which differed from those commonly seen here, inasmuch as its body was perfectly diaphonous before feeding, and even after this operation had been completed the transparency was such as would have made it a splendid medium for observing *in situ* any changes going on within, had it been possible to bring live specimens thus far. I am not able, therefore, to say, from personal observation of its habits, that it is hostile to embryo development, though analogy and the marked absence of filarially infested persons, or disease attributable to the parasite, tends strongly to suggest that it may also be assumed incapable of playing the part of nurse.

I now proceed to give the measurements and description of the only varieties of mosquito I have been able to discover, striving, by the accompanying sketches, to facilitate reference

and recognition. For comparative purposes, similar descriptions and sketches of what I will call the true filaria-nurturing mosquito, as got from Amoy, are also given.

By far the commonest Formosan mosquito is the striped, or "Tiger" variety (fig. 1). The appearance, to which its name is due, is caused by a series of black and white stripes on the second and third pair of legs, thorax, and proboscis of the insect, while in further justification of the appellation, most formidable claws, or, indeed, talons, arm the extremities of the middle and posterior legs. At base of proboscis are a pair of palps, with double antennæ, plumose in the male, and pilose in the female. The proboscis striped, of equal diameter throughout, has pointed extremity, through which may be seen several lancet-shaped spicules protruding. The wings, dark, transparent, with delicate reticulated venation, to which are attached, generally from one side only, lanceolate appendages, or winglets, which also form a fringe or periphery of wing.

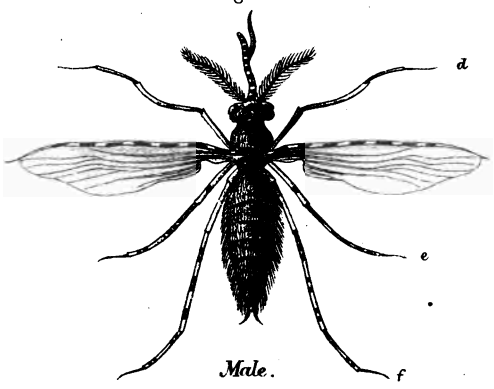
Measurements:—Head and proboscis, $\frac{8}{96}''$; proboscis alone, $\frac{6}{96}''$; wings, $\frac{10}{96}''$. Legs: 1st pair, 3-jointed; 2nd pair, 2-jointed, $\frac{28}{96}''$; 3rd pair, 5-jointed, $\frac{32}{96}''$; antennæ, $\frac{6}{96}''$.

Another not uncommon variety, on account of its colour, may at first sight be mistaken for the filarial-nurturing species, but by closer investigation it can be easily distinguished. This mosquito (fig. 2) is light brown all over, save on the back, the central part of which is covered with dark scales. The posterior leg has two short claws, and is smoother than the middle one, which latter, however, is without claws, though covered with rough hair-like processes. The proboscis is distinctly hirsute from base to tip, of equal diameter throughout, tapering to a point at end. At base of proboscis are two short abrupt palps, and a pair of antennæ having long hairs arranged in sets of four at equal distances from each other. Wing, similar to those of "tiger" variety, though somewhat darker.

The following measurements were taken:—body (thorax and abdomen), $\frac{16}{96}''$; head, $\frac{2}{96}''$; proboscis, $\frac{6}{96}''$; antennæ, $\frac{8}{96}''$.

The third and last variety I have been able to differentiate, is given more on account of its remarkable peculiarities than for anything else. This insect I will call the "Ejecting" mosquito, from its habit of ejecting at anal extremity the blood it draws in while feeding. Discharge and suction seem to go on simultaneously, for as soon as one drop falls off, another commences to form, which in turn gives place to its successor. This process has been observed to go on for five minutes consecutively, without any apparent increase of

Fig. 1.



Male.

COMMON TAKOW MOSQUITO.
"TIGER" MOSQUITO.

BROWN MOSQUITO.
found in Takow.

Ventral aspect.

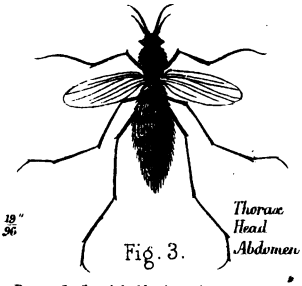
Light brown all over save central part of back which is covered with series of dark scales.

Fig. 2. *Thorax & abdomen* $\frac{8}{48}$" *long.* *Head* $\frac{1}{48}$"

Brown head and tip of abdomen, intervening part covered with black scales.

Dorsal aspect.

EJECTING MOSQUITO.



Body $\frac{19}{96}$"

Thorax $\frac{3}{96}$"
Head $\frac{5}{96}$"
Abdomen $\frac{14}{96}$"

Fig. 3.

Brown body with black stripes on back and 6 black spots on each side of brown abdomen.

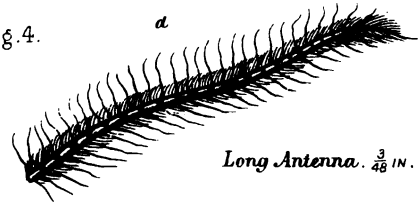
Tip of Proboscis showing bulbous extremity

$\frac{4}{48}$ IN.

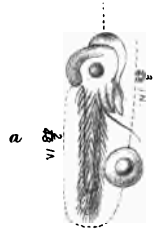
Wing of filaria nurturing Mosquito.

FILARIA NURTURING MOSQUITO.

Fig. 4.



Long Antenna. $\frac{3}{48}$ IN.



Head and Proboscis with single palp.

bulk in the mosquito. The blood is certainly changed during its passage through the alimentary canal, for the fluid discharged, though red in colour, is much more serous, and does not clot. On microscopic examination the colour is seen to be due to disintegrated and shrivelled corpuscles, with some granular *débris*, probably also the remains of broken down blood-globules. This peculiarity would seem at once to stamp the insect as physically incapable of acting as filarial-host, but may still be deemed of sufficient interest to render a more detailed description acceptable. The whole insect is brown (fig. 3), with black lines on back. On the belly six black spots are symmetrically arranged on each side of median line. The proboscis is marked like that of the "tiger" variety with three broad bands, one black at base and tip respectively, and an intervening uncoloured one; it is smooth, and of equal calibre throughout, with two long straight antennæ. The anterior leg has three joints; the middle leg has four joints; the posterior leg has five joints; the body measures, $\frac{12}{96}''$; thorax, $\frac{3}{96}''$; abdomen, $\frac{14}{96}''$; head, $\frac{2}{96}''$; proboscis, $\frac{7}{96}''$; I may add that this mosquito is not very common, and seems to be confined to a limited area in Takow.

The foregoing then are all the varieties I have been able to distinguish as yet in South Formosa.

I feel that these descriptions are by no means complete; but perhaps as the distinctions given are at least well marked, and offer readily recognisable points of difference, they may serve as incentives to that further and more minute examination which others, resident in places where the filarial-nurse insect is found, may be prompted to undertake.

The following is the result of repeated examinations of the filarial mosquito obtained from Amoy, and which may also prove useful in initiating a more minute and detailed description by those possessing the improved facilities for making such, which fellow-residence affords.

Filarial Mosquito.—A dark brown body, which is evidently smaller than any variety I have found here. Posterior leg (fig. 4), covered with delicate leaf-like appendages, no claws. At base of proboscis are two antennæ, beaded, with filamentary processes coming off at each joint, one shorter than the other, the bead-like joints of former larger than those of latter, while at each constriction there are only two filaments, one on each side. Short abrupt palps. The proboscis itself terminates in a cone-like bulb, markedly differentiating the insect from those previously described.

Wings, ribbed, covered with obtuse leaf-like appendages, arranged feather-wise on each rib, and round the periphery.

The following are the measurements taken: head, $\frac{2}{96}''$; proboscis, $\frac{4}{96}''$; wings from base to tip, $\frac{8}{96}''$; long posterior legs, $\frac{22}{96}''$; long antennæ, $\frac{6}{96}''$; short antennæ, $\frac{5}{96}''$. Below is a table of comparative measurements got from the South Formosan varieties, and the Amoy filarial musquito, showing the parts distinctly different in each species.

Comparative Measurements of South Formosan Mosquitos and of the Filaria-nurturing Insect from Amoy.

	South Formosan Varieties.			Amoy Varieties.
Parts.	Tiger Mosqto.	Brown Mosquito.	Ejecting Mosquito.	Filarial-nurturing or Amoy Mosquito.
	Inch.	Inch.	Inch.	Inch.
Head and Proboscis	$\frac{8}{96}$	...	...	$\frac{6}{96}$
Head	$\frac{2}{96}$	$\frac{2}{96}$	$\frac{2}{96}$	$\frac{2}{96}$
Proboscis.....	$\frac{6}{96}$	$\frac{6}{96}$	$\frac{7}{96}$	$\frac{4}{96}$
First pair Legs	...	...	...	...
Second pair Legs ...	$\frac{28}{96}$	...	...	...
Third pair Legs.....	$\frac{30}{96}$	...	...	$\frac{22}{96}$
Antennæ	$\frac{8}{96}$	$\frac{8}{96}$	...	long $\frac{6}{96}$ short $\frac{5}{96}$ } Ant.
Wings	..	...	...	$\frac{8}{96}$
Body.....	...	$\frac{16}{96}$ thorax & abdomen	$\frac{19}{96}$ (whole)	...
Thorax.....	...	...	$\frac{3}{96}$	...
Abdomen	...	...	$\frac{14}{96}$	...

From these, and other observations, I feel justified in reasserting that the filarial mosquito, or at least that species which acts as such on the mainland, is absent from the south part of this island; while, judging from the absence of filarial disease all over Formosa, I venture to submit that this all-essential intermediary, for some reason—as yet unknown, cannot, or does not, exist at all in the island. People are constantly coming and going from Amoy, water tanks arrive every day, but still the closest search has failed to discover a trace of the Amoy insect anywhere in the south half of Formosa.

I also arrange descriptions of the four varieties of mosquitos in parallel columns to further facilitate comparison.

South Formosan.			Amoy.
First.	Second.	Third.	Special.
Tiger Mosquito.	Brown Mosquito.	Ejecting Mosquito	Filarial Mosquito.
Striped black and white on body, proboscis, second and third pair of legs; proboscis of equal calibre throughout; middle and posterior legs with claws; two palpi; two antennæ, plumose or pilose; wings clear, transparent, ribbed, with lanceolate winglets, or appendages attached to ribs and round periphery of wings.	Light brown body and head; back covered with dark scales; posterior leg with two short claws, middle, hirsute; proboscis of equal calibre throughout; two short abrupt palpi; two antennæ with filamentary processes arranged in fours; dusky wings, ribbed, with few winglets or appendages attached to ribs and round periphery of wings.	Brown body with black lines on back, and black spots on belly symmetrically arranged on each side of median line; proboscis straight and of equal calibre, black and white stripes or bands; two long straight naked antennæ; short palpi.	Body dark brown, generally smaller than other varieties; legs unclawed, covered with obtuse, leaflike appendages similar to those on wings; proboscis terminates in cone-like bulb; two antennæ of different lengths, beaded or distinctly jointed; pilose, the longer with single long filaments springing from each constriction at joints; shorter, with longer joints than its fellow; wings ribbed, with delicate leaflets arranged featherwise on each side of ribs, and fringing periphery of wings.

Before going on further to describe the investigations made to discover the ultimate destination of embryo-filaria remaining in the blood of the human host, it may be as well here to notice the leading objections raised by those who favoured my last paper with their criticism.

Dr. Manson, and to some extent Dr. Cobbold, thought the great difficulty in accepting my views was that they seemed to suggest periodic, or intermittent reproduction on the part of the parent worm, and, indeed, the former made this almost the sole ground of dissent. Assuredly, if we take it for granted that all the embryos in the body during the hours they appear in the blood are contained in the vascular system, then if these are destroyed their place can only be filled by a new swarm, but knowing as I did, what Dr. Manson also calls attention to, that, in cases of chyluria, and lymph scrotum, a continuous outcome of embryos is kept up during the whole day, I did not intend it to be understood that the supply was intermittent, or absorbed as soon as provided. I did not discuss this part of the subject at length, because more occupied with the question as to whether the embryos in the blood died there, or betook themselves to some resting place. From what I submit hereafter, however, it will be seen that the suggestions I then offered, and those I now present, as to the filarial reproduction and existence in the body of primary host, are not inconsistent with each other, and that neither intermittence of production, nor impro-

bable enormity of swarm, are essential to the views put forth.

I was not fortunate enough to obtain Dr. Manson's corroboration of my experiments as to the longevity and vitality of the embryo at the different hours of its existence in the blood, but I was very gratified to find that Dr. Stephen Mackenzie, in his kindly review of my paper for the *London Medical Record*, January 1882, p. 5, stated he had repeated my experiments, and was able to confirm them. From this, and the fact of three other observers (two of them medical men) having checked my observations on the spot, and agreed with me, I may hope that some accidental circumstance interfered with the observations made by Dr. Manson, and perhaps prevented his arriving at similar results to those got in London and Formosa. Having verified, in so far as possible, the experiment and observations previously detailed, I have devoted most of my efforts towards determining the question left unsettled as to the ultimate destination of the filariæ not abstracted by the mosquito.

In considering at the offset the data we have to work on, there is one fact which seems to me to stand forth very prominently, and yet has not, so far as I know, been dwelt on to the extent its value as a preliminary factor in clearing the way for further investigation seems to warrant. I allude to the *absolute* need there seems to be for the *regular* and *periodical* removal of certain numbers of the embryos, so as to make room for those constantly coming into existence, and which must, in turn, have their chance of mosquito-deliverance. In other words it seems very certain that the unremoved filariæ must go somewhere whence their return to the blood is impossible. Assuming that parturition in the parent is continual, at the lowest estimate a single worm should produce about two million embryos in the twenty-four hours, for Dr. Manson tells us that the minute filaria corvi torquati was seen to give birth to "twenty or thirty embryos every few seconds".* If we suppose this number to be brought forth each three seconds, that would give a total of 864,000 in the twenty-four hours, or say in round numbers, one million.

In estimating the number of filariæ present at one time in the blood of a man, the difficulties are very great, and indeed only an approximate result can be hoped for. To arrive at this, let us assume an adult weighing, say, twelve stone, and therefore possessing about fifteen pounds of blood; let us suppose the amount that goes on a slide to be about a minim,

* *Medical Reports*, No. xxiii, p. 13.

and that the total quantity of blood in his body would be about 115,200 minims. With a view to getting some idea of the number of filariæ on a slide, *i.e.*, in a minim of blood, I have gone over the records of filarial finds at different hours and for several days; taking the days and hours when the maximum and minimum number of embryos were present, and striking an average, I have been able to arrange a series of 100 records, covering several days for each individual. I should mention that I have utilised the Customs Reports and other publications for this purpose. From these the average number of filariæ found in one minim was thirteen. On this basis there would be 1,427,600 filariæ present in the whole vascular system, but to allow margin let us put them at one million and a half.

Now, if our calculation as to the daily produce of the minute filaria corvi torquati be tolerably near the mark, it will not be over-estimating the capacity of the Filaria Sanguinis Hominis to assume the daily produce as being close on two millions. Dr. Manson* calculates the number of filariæ present at one time in an infested man at "*more* than two millions"; but does not say how he comes at this result. For our present purpose I propose taking the figures I have given. By the calculation then, the parent worm would produce about half a million more filariæ each day than appears in the blood. Further on, when I submit my views as to the phases of existence passed by the embryos in the human body, I will offer suggestions as to why this is, and how it accords with the requirements of the parasite, and adjustments of nature.

Even supposing exactly the same number were produced as are seen in the hæmic circulation, and that the new swarm only entered the blood every twelve hours, it is obvious that at this rate, unless a very considerable outlet were provided, a block must soon ensue. It is clear that even under the most favourable circumstances mosquito aid would be comparatively useless; then what becomes of the unrescued residuum? It is highly improbable the embryos can re-enter the lymphatic system, the valvular folds at the entrance of the thoracic duct would bar retreat by that opening, to say nothing of other forces hostile to such an attempt; nor does it seem probable that, like the white corpuscle, they can make their way through the capillary coats. Even if they could, however, the evils of constant advent, with no relieving means of withdrawal, would be just as conspicuous and urgent. They do not rest in the organs, as Dr. Manson at

* *Customs Medical Reports*, No. xiv, p. 9.

first thought might be the case, for he tells us,* that blood aspirated from the spleens of two filarious patients, and also that coughed up by another, during the day, contained no filariæ. I am also in a position to affirm the same of splenic blood, with the addition of like negative results obtained from several liver aspirations. But even in the absence of these "proofs by exclusion" it seems to me that the physical obstacles to anything short of complete removal from the economy, giving space to the crowds fast collecting, must be inadequate, and therefore that all suggestions involving accumulation, such as temporary stay in this or that place, must be put aside as inapplicable to the obvious requirements of the case.

It may be right here to mention that my observations, and I have tried to devise special experiments for the purpose, tend to make me question very much whether the embryo has any inherent power of locomotion. No doubt there is very vigorous action, but this seems to be all directed towards the centre of the circle formed by the parasite, and although I have placed it in positions favourable to freedom of motion, such as between comparatively separated glasses, adding fluid of serum-density so as to cause the blood-corpuscles to run about freely, the embryo losing none of its vigour but stretching itself out at times as though to attach red globules, I could not detect the slightest onward progress; I have put the blood in glass capillary tubes, where one would expect locomotary powers to show themselves if present, but such has not been the case; and I have seen the same creature on the field of my microscope, when making observations on the solution question, for days, including the time when its full vigour ought to have enabled it to move, if at any time competent to do so. In fact, the earliest occasion on which I have seen the embryo spontaneously moving, was after twenty-four hours' stay in the mosquito, and subsequent to shedding its integument. In the human blood-vessel I believe it is helplessly borne along by the force of the current, and this, if it is as I suspect, may be thought to have an important bearing on any question dependent on the autonomous movements of the embryo after it has got into the vascular system. That it has some slight holding power I think is obvious, and I have tested this by means of currents of different strengths; indeed, from what I saw, I do not doubt but that in the slowly flowing lymph-current it could easily control progress, though I think not aid it, but the blood-flow certainly seems quite beyond all its powers of

* *Customs Medical Reports*, No. xxiii, p. 8.

resistance. Granting that the liquor sanguinis is capable of dissolving the dead filarial embryo, and holding that those parasites not extracted by the mosquito during the night die, and so make room for their successors of the following evening, the question still remains: What is it that brings about this mortality? As Dr. Manson pointed out it is not something connected with the mere state of rest, or wakefulness, as for several hours before the host goes to sleep the embryos are disporting themselves in his blood; the attraction must therefore exist in peculiar conditions of that fluid at these times, so must their disappearance or death be consequent on the hæmic state during the period of embryo absence. Dr. Manson suggested one or two theories, such as diurnal magnetic influences, barometrical pressure, variations in temperatures, etc.; and made several experiments and observations to test these suppositions, with the result that he felt constrained to abandon his surmises. Dr. Mortimer-Granville seems to me to have struck the key-note at once, when he pointed out, on hearing of Dr. Manson's discovery as to the periodicity of filarial presence in the blood, the probability of this being due to physical changes and conditions peculiar to each period. Following and supporting this conjecture, came Dr. Stephen Mackenzie's brilliant and ingenious experiment, whereby he proved that reversing the hours of sleep also altered those of embryonic advent to the circulation. With a wisdom that follows "a prompt", we see this change to be nothing more than must necessarily have taken place as the subject of Dr. Mackenzie's observations travelled from India to London.

To illustrate what I mean, I have drawn up the following Table, showing the various alterations in time at different points of call on the journey home, and consequently in the hours of filarial movements. Taking the difference in time between Greenwich, and the chief places of each Indian Presidency, I find this to be as follows:—Calcutta, 5 hrs. 53 mins. 20 secs.; Bombay, 4 hrs. 51 mins. 12 secs.; Madras, 5 hrs. 20 secs. 59 min.; the mean difference between the Indian shores and London would be about 5 hrs. 21 mins. 50 secs. In ignorance of the actual port of embarkation selected by the patient, I have to adopt this approximate method, which moreover will serve our present purpose equally well. The Table shows the various changes undergone.

Table showing variations in time of filarial appearance, on a voyage from India to London.

	H.	M.	S.
Difference of time between Greenwich, and the port of embarkation in India	5	21	50
Colombo	5	19	23
Aden	3	0	10
Port Said	2	9	17
Gibraltar	0	21	24

Supposing filariæ appeared in blood at 7 p.m, mean time, in India, this hour corresponds to mean time at:

Colombo	6	57	33
Aden	4	38	20
Port Said	3	47	27
Gibraltar	1	59	34
London	1	38	10

Reckoning by Indian mean time, therefore, the filariæ postpone their appearance at:

Colombo ... ... For	0	2	27
Aden ... ... „	2	21	40
Port Said ... ... „	3	12	33
Gibraltar ... ... „	5	0	26
London ... ... „	5	21	50

To make this plainer, let us imagine that a filarious Fijian, from Voona Point, Faviuni, one of the Fiji group, and situated in 170° 56′ west longitude, makes a voyage to London, he will, on arrival, have exactly accomplished what Dr. Mackenzie did in the hospital wards. That is to say, the hours his blood contains embryos in England are exactly the opposite of those during which the parasites were present in his circulation while at Faviuni.

But neither Dr. Mackenzie's experiment, nor the geographical exemplification of it he has taught us to deduce, owes its present chief interest to the mere curiosity of the facts disclosed; indeed, to my thinking, by his ingenious thought, and skilful accomplishment of the idea, we are put in possession of one of the most important clues towards the solution of the ultimate-destination-problem, that has been gained since Manson made his mosquito-nurture, and periodicity observations. I say this advisedly, for we are now in a position to go a step further than Dr. Mortimer-Granville, in assuming the influences so strangely affecting the embryos to be regular and physiological ones, intimately connected with the systemic routine. Then by following out the hint thus given us, we may find some other and more familiar phenomena which owe their existence to similar

causes, and these traced out may perchance lead us to the solution desired.

In the variations of body temperature it would seem that we have an analogous, or somewhat approximate, phenomenon. Thus, it changes periodically, and after a more or less regular order. No doubt the same variations take place in travelling from one part of the world to another, and what is a fixed morning and evening record in one latitude, becomes quite changed with locality and other co-incident circumstances.

In passing, I might here suggest to those of my brethren who go down to the sea in ships, whether naval or commercial, that on their various voyages, to and from different quarters of the globe, careful registers of the temperature curves, as the ship changed her position day-by-day, would be highly interesting, and might be productive of considerable results.

To return, we are certainly in possession of the physical causes affecting body temperature, and although we have seen that thermic variations themselves, have little or no effect on the embryo periodicity with which they are coincident, still it appeared to me that possibly some of the chief *causes* influencing animal heat, may not improbably be those that act on filarial wanderings. Carrying out this idea, and utilising Dr. Mackenzie's discovery as a hint for further investigation, in my preliminary pursuit, through analogy, I have adopted that gentleman's method of "turning night into day", with a view of seeing the effect this had on the usual temperature record. No doubt the following tables only depict what geographical observations must have frequently shown; but for the purposes at present interesting us, I have thought it best to make confirmatory experiments.

Finding it difficult, if not impossible, to get adult native subjects to submit to the restraint and monotony entailed by the necessities of this experiment, I would have had to give up all idea of carrying out my project, but for the zeal and willingness of my two little daughters, aged eleven and twelve years respectively. Being able to rely both on their intelligence and *bonâ fides*, I was glad to avail myself of their volunteered aid, and on the fourth of January of this year the observations commenced, extending over twenty-four days, during the whole of which time the young people stuck loyally and steadfastly to their undertaking. The mean temperature of the air was about 60° Fahr.

TABLE NO. I.

Records of temperature taken from H. M., *æt. twelve years, during two periods of eight days, up in daytime and sleeping at night; also of a similar period of eight days, wakeful at night and sleeping during day. Between each period a day is allowed for the change of habit.*

Up during Day and sleeping at Night.		Wakeful at Night and sleeping during Day.		Up during Day and sleeping at Night.	
	Degs. Fahr.		Degs. Fahr.		Degs. Fahr.
1st day.	6 a.m. ... 96.	10th day.	96.20	19th day.	94.80
	12 „ ... 99.40		99.		97.20
	6 p.m. ... 99.60		95.		99.
	12 „ ... 98.		95.		95.
2nd day.	6 a.m. ... 98.	11th day.	95.20	20th day.	95.20
	12 „ ... 99.		95.20		100.20
	6 p.m. ... 100.		95.60		98.80
	12 „ ... 98.		96.20		98.50
3rd day.	6 a.m. ... 96.40	12th day.	96.20	21st day.	96.20
	12 „ ... 100.		98.20		100.
	6 p.m. ... 100.20		94.90		100.
	12 „ ... 98.50		96.80		98.40
4th day.	6 a.m. ... 96.	13th day.	97.40	22nd day.	94.20
	12 „ ... 100.		97.20		98.70
	6 p.m. ... 100.		96.10		100.60
	12 „ ... 98.50		98.50		100.20
5th day.	6 a.m. ... —	14th day.	97.	23rd day.	95.60
	12 „ ... 98.20		95.80		100.60
	6 p.m. ... 99.80		95.80		100.
	12 „ ... —		98.20		98.
6th day.	6 a.m. ... 96.80	15th day.	98.20	24th day.	94.80
	12 „ ... 97.		97.80		96.80
	6 p.m. ... 98.50		96.80		98.
	12 „ ... 98.50		95.20		97.80
7th day.	6 a.m. ... 96.	16th day.	98.80		
	12 „ ... 90.		97.40		
	6 p.m. ... 97.20		94.60		
	12 „ ... 98.		97.20		
8th day.	6 a.m. ... 96.	17th day.	95.60		
	12 „ ... 97.		96.40		
	6 p.m. ... 97.		94.40		
	12 „ ... —		96.20		
9th day.	6 a.m. ... 97.	18th day.	94.20		
	12 „ ... 97.		98.50		
	6 p.m. ... 97.		97.40		
	12 „ ... 95.20		97.80		
MEANS.	6 a.m. ... 96.52	MEANS.	96.82	N.B. — These means include the whole 15 days of day wakefulness and night sleep.	
	12 „ ... 98.57		97.13		
	6 p.m. ... 98.79		95.40		
	12 „ ... 98.25		96.68		

TABLE No. II.

Records of temperature taken from A. M., æt. eleven years, during two periods of eight days, up in daytime, sleeping at night; and also of a similar period of eight days, wakeful at night and sleeping during day. Between each period a day is allowed for change of habit.

Up during Day and sleeping at Night.			Wakeful at Night and sleeping during Day.		Up during Day and sleeping at Night.	
		Degs. Fahr.		Degs. Fahr.		Degs. Fahr.
1st day.	6 a.m.	96.60	10th day.	97.	19th day.	95.40
	12 „	98.50		96.		98.60
	6 p.m.	98.50		95.40		98.60
	12 „	—		98.50		95.20
2nd day.	6 a.m.	97.	11th day.	98.50	20th day.	95.60
	12 „	98.50		96.		98.
	6 p.m.	98.		96.20		98.20
	12 „	97.		98.		97.
3rd day.	6 a.m.	96.40	12th day.	97.20	21st day.	94.80
	12 „	98.50		99.20		98.20
	6 p.m.	98.70		97.20		98.50
	12 „	98.		97.20		98.
4th day.	6 a.m.	96.80	13th day.	97.20	22nd day.	95.40
	12 „	98.60		98.20		98.50
	6 p.m.	99.10		99.		99.
	12 „	—		98.60		98.
5th day.	6 a.m.	—	14th day.	97.20	23rd day.	94.40
	12 „	97.60		97.80		98.60
	6 p.m.	99.40		96.		99.
	12 „	—		98.20		98.50
6th day.	6 a.m.	96.40	15th day.	98.20	24th day.	95.20
	12 „	98.		99.		98.80
	6 p.m.	98.50		96.26		98.60
	12 „	97.		95.60		97.
7th day.	6 a.m.	96.20	16th day.	98.20		
	12 „	99.		93.60		
	6 p.m.	98.70		95.80		
	12 „	97.		96.80		
8th day.	6 a.m.	97.40	17th day.	96.10		
	12 „	99.		96.40		
	6 p.m.	98.50		94.40		
	12 „	—		97.		
9th day.	6 a.m.	96.	18th day.	94.60		
	12 „	98.50		97.60		
	6 p.m.	94.40		98.70		
	12 „	95.80		99.60		
MEANS.	6 a.m.	96.60	MEANS.	97.45	N.B.—Means for day wakefulness and night sleeping include all 15 days.	
	12 „	98.47		97.28		
	6 p.m.	98.42		96.28		
	12 „	97.25		97.46		

On referring to Tables Nos. I and II, it will be seen that as soon as nocturnal wakefulness began, the relative heights of the morning and evening records were reversed, as were those for other hours. Thus the morning became the highest for the period and the evening the lowest. The Table of Means shows this best. While the children were keeping up at night, the general rate of the nocturnal readings were much lower than those to which they were supposed to be analogous; but against this we may perhaps set off the fact of the production of carbonic acid during the night, as compared with that produced during daylight, being in the proportion of *one* of the *former*, to *one and a quarter* of the *latter*, and this is said to be quite irrespective of sleep or wakefulness. Accepting this as evidence of so much heat generated, we may expect to find the nocturnal manifestation of the latter lower by *just* that proportion of constant difference, even though other circumstances were artificially assimilated to those of diurnal occurrence. This does not account, however, for some extremely low indications given by night and day, and especially during the latter time, in both children. Thus, with H. M., 96° was registered during the day-time; and with A. M., 96° 20′; besides this, in H. M.'s record we also find 96° 40′; 96° 80′; and in A. M.'s the same. On the day the habit was changed, during which they slept as much as possible, H. M.'s lowest record was 95° 20′, and A. M.'s 95° 80′; in the nights, while wakeful, H. M.'s, and A. M.'s lowest was 94° 40′, on the same night and hour (17th day); H. M., during this time also recorded 95° (twice), 95° 20′ (thrice), 95° 60′, 95° 80′ (twice), and A. M., 95° 40′, 95° 60′ (twice), besides other abnormally low readings. On the day of change to the normal routine A. M.'s lowest was 94° 60′, H. M.'s, 94° 20′. On getting back to night rest, H. M. had 94° 20′ (once), and 94° 80′ (twice), besides 95° 20′, and 95° 60′, 96° 20′, at other times. A. M.'s lowest was 94° 40′, then 94° 80′, 95° 40′ (twice), 95° 20′ (thrice), and 25° 60′, at other times. These low temperatures no less surprised than alarmed me at first, but I could detect nothing to warrant anxiety. The children are strong, well developed and nourished; they appeared then to be, and have been since, in perfect health, eat well, are active and lively, and complained of no subjective sensations in keeping with the mercurial indications. On every occasion these observations were taken with three different instruments, one of which had the Kew correction.

Dr. Aitken, in his work on Practice of Medicine,* states

* Sixth edition, vol. i, p. 247

that Mr. Jas. P. Cassels of Glasgow was "much struck", while using the thermometer on children completely recovered from measles, by the low records he got; and in a Table of temperature records taken from a child sixteen and a half months old, "*when asleep*, and every source of error carefully avoided", he shows results "*below* those of adult life". He took six records as follows: "96° 2′, 96° 4′, 96° 4′, 97° 2′, 97° 6′, 97° 4′ Fahr."

I have a strong impression of having noticed, in some of the medical publications, communications calling attention to this peculiarity in children, but cannot at the moment recall when or where. At any rate it is a fact well worth bearing in mind, and may save much of the anxiety likely to arise if only discovered at the bedside by one unprepared for such results.

Now, looking to our immediate object, we know that the variations in temperature are principally due to chemical changes going on in the body, and may be, at present, considered only in so far as these concern the access of oxygen, and production, or exhalation, of carbonic acid. These, at any rate, are undoubtedly the chief and most marked differences found between the conditions of the blood at the two periods under review. Turning to the embryo filaria, we find that an anxiety to possess itself of oxygen seems to be a very conspicuous and constant trait in its disposition. Thus, it will be remembered that on the slide, if the animal happens to get washed away from the bulk of the red corpuscles, it becomes most restless, at once stretching out its body, as though seeking for something. On coming in contact with the globules it eagerly embraces them, rolling these over and around its body, and, if washed back to the general mass, these motions continue for some time. After a little, however, as if having recovered some of the vitality lost during removal from corpuscular contact, it resumes its vigorous movements and the semi-oval form that seems to be its normal and healthy position. Again, it will be recollected that the embryo leaves the human circulation and enters the mosquito prior to the blood of its primary host undergoing the changes which are suggested bring about its death. Now, in the mosquito, the condition certainly changes, but in a direction *favourable* to development, and which the instinctive desire of the parasite seems to indicate, that is to say, the continued and regular supply of oxygen is secured. Thus, we may put it, the embryo having stayed as long as is compatible with its safety and comfort in the human host,

changes its abode for one where these essentials approximate or exceed those present in man's blood during the night. Even here, it would seem, the embryos display an elective ability, selecting a position where the most oxygen is available, and where there is less exposure to the products of oxidation; for, as Lewis has pointed out, the imbibed filariæ soon emigrate from the abdomen of the mosquito to its thorax. Dr. Manson fully confirms this statement.

Considering the circulatory structure of insecta, we find that the dorsal vessel, or rudimentary heart, discharges its contents into the neighbourhood of the head, and so those filariæ, situated high up in the body, are being constantly bathed by a fluid richly charged with oxygen. True, if lying in the abdominal cavity, they are not deprived of this gas, for the tracheæ or spiracles would no doubt keep them more or less supplied; but, for obvious reasons, the conditions of the thoracic position are superior, and calculated to afford all that is required for sustenance and development in more concentrated and convenient form. At least, this seems to me a probable explanation of this selective tendency in the parasite, while I think it proves the constant need for and seeking after oxygen, a point more specially under notice at present.

I take the following from Carpenter's *Physiology* (7th ed., p. 343), where, referring to the varying amounts of carbonic acid exhaled during day and night, he says: "Sleep or watchfulness. The amount of carbonic acid exhaled during sleep is considerably less than that set free in the waking state. This is particularly shown by the experiments of Scharling. . . . . Thus in one case the hourly exhalation sank from 160 to 100, and in another from 194.7 to 122.3." On page 344: "From the experiments of Scharling on the human subject, it would appear that the average proportion exhaled by day, to that exhaled by night, is as one and a quarter to one; and this difference does not seem to be affected by sleep or wakefulness. . . . . There was the least during the middle hours of the night, a slight increase with sunlight, a large increase after meals, and a decrease before them, and a prolonged and inevitable fall after about 9 P.M." Compare this with the periods of filarial appearances in varying numbers, and their complete withdrawal, when a very striking relation seems indicated. On page 345, the author gives the result of Petenkofer and Voit's experiments on a healthy man in the following table:—

Period of the day.	Elimination of CO_2, HO_2 through Skin and Lungs in Grammes.		Amount of Oxygen absorbed.	Percentage of the Inspired Oxygen in the Carbonic Acid.
DAY.				
6 a.m. to 6 p.m. ...	532.9	344.4	234.6	178
NIGHT.				
6 p.m. to 6 a.m. ...	373.6	483.8	474.3	58
Total in 24 hours ...	911.5	828	708.9	94
A few days later the same man worked till exhausted.				
DAY	884.6	1004.8	294.8	218
NIGHT	399.6	947.3	659.7	44
Total	1284.2	2042.1	954.5	98

He goes on to say, page 346: "This table shows a remarkable excess of the diurnal against the nocturnal elimination of carbonic acid, especially after work, and a corresponding increase in the percentage of the absorbed oxygen which is thus discharged." It is to be noted at present, therefore, that according to these authorities the amount of absorbed oxygen discharged during the night is as 58 to 175 of that eliminated as carbonic acid in the day for a man undergoing ordinary labour, while for the same man working hard, as most Asiatics do, the proportion is as 44 to 218. Allowing for the amount combining with hydrogen, the blood must still be markedly richer in free oxygen at night than during the day. On page 223 (*op. cit.*) it is stated that Sczelkow and Bernard found that venous blood, returning from muscles at rest, contained on the average 6.71 per cent. *more* carbonic acid and 9 per cent. *less* oxygen than arterial blood, while from muscles in action the excess of carbonic acid was 10.79 per cent., and the deficit of oxygen 12 to 16 per cent. in venous blood as compared with arterial. Considering these facts, it seemed to me possible that the embryos were either killed by carbonic acid acting as a direct poison, or, by excluding oxygen, it indirectly brought about the same result. To decide this question I naturally made observations on the direct application of carbonic acid to the blood, and in order to get a sufficient supply of the latter the median basilic vein was punctured. I was at once struck with the marked difference in vigour and vitality shown by the embryos in this blood, as compared with those in a drop extracted from the finger at the same time by the ordinary method. Proceeding to test the longevity of the filariæ in each specimen of blood, two thoroughly oiled slides were rapidly applied to

the puncture in the vein, so as to avoid as far as possible oxygenation taking place, and the cover quickly slipped on. These precautions were deemed advisable, as I observed that if the drop of venous blood be exposed for a little time to air, the filariæ distinctly became brisker, and exhibited something of their normal activity, though still comparing unfavourably with their fellows taken from the capillaries. The following are notes of the results obtained in January 1884: "*Drop of blood from median basilic vein. Eight embryos on slide.* Marked difference in vigour from those in blood drawn by prick from finger, all more or less feeble, and stretched out. By the third day none were to be seen on slide, this being a somewhat quicker result than was got with the filariæ withdrawn from arterial circulation in the morning.* Those extracted in the usual way, from the finger, besides being much brisker from the first, and preserving their vitality much longer, had not all disappeared from the slide until the morning of the ninth day."

Experiment No. 2.—Three ordinary soda-water bottles were taken, and about an ounce of venous blood allowed to flow into each. Once more taking the mean of five slides charged with some of this blood, I calculated that each minim contained about six filariæ, which would give for the whole quantity 8,640. Prior to covering the slides the drop was freely exposed to the air, with the effect of considerably reviving the otherwise debilitated embryos. These were put aside and examined daily, and by the fourth day all the filariæ on the different slides had disappeared. To return to the bottles. No. 1 was placed under the gas-pump, and well-washed carbonic acid was forced in at the ordinary pressure used for soda-water, viz., eight atmospheres. No. 2 was similarly treated, save that the gas only had a pressure of five atmospheres; and with No. 3 the gas was merely allowed to flow in, at a pressure of about five pounds on the square inch. This, of course, immediately produced on the blood the usual effects of contact with carbonic acid. The bottles were securely corked in the ordinary way by the machine, and put aside for twelve hours. On opening the first and and second, though several slides were examined from each, and the blood taken at different depths, I was only able to discover on one slide, from No. 1 bottle, the REMAINS of three filariæ. From No. 2 I found two dead embryos on one slide and some remains on the others. With No. 3 I got on one slide five very feeble filariæ, on another three, and on a third four and remains of two others. We must discard Nos. 1

* See former paper, *Customs Medical Report*, No. xxi (1881), p. 17.

and 2, as probably the great pressure at which the gas was forced in took the chief part in causing the death of the parasites, though I am of opinion that the carbonic acid certainly hastened the solution. With No. 3 it is quite possible that the carbonic acid may have brought about the result, but I could not form any decided opinion, from even this experiment, as to whether the carbonic acid acted as a poison, or merely destroyed life by the exclusion of oxygen. As may be supposed, I had considerable difficulty in persuading To-ah to submit to an operation that appeared so formidable to one of a race who gauge the severity of all injuries by the amount of blood-flow. By dint of ample reward, however, and assurances that I had taken every means to convince myself of the harmlessness of the procedure, I got him, for this occasion only, to submit as I have described. This occurred eighteen months ago, and I may mention that he is in perfect health, allowing me at intervals to tap his finger by needle-prick; still, even for this, he is by no means so complaisant as formerly. I mention this so that the experiments, unrepeated as they were, may be taken for what they are worth; and, indeed, all through, the fact of being so heavily handicapped by being confined to one individual, the embryos in whom were palpably getting less numerous, renders all that I have done, of late years at least, and which are dependent on observations made with a solitary case, in verity only valuable so far as they seem to foreshadow what may be borne out by investigations which must be much more extensive and searching before that scientific accuracy can be arrived at, so essential to the deduction of positive conclusions.

The following appears to lend some support to the idea that, *in the presence of an excessive supply of oxygen*, the embryos are more indifferent to the action of carbonic acid (Exp. No. 3). Through a drop of blood on a slide I passed a continuous current of carbonic acid for some considerable time. The cover glass was then put on and the slide preserved in the usual way. I was not able to convince myself that the embryos were materially affected either during the time the gas was playing on them, or afterwards, as to their longevity. I should state that the blood so acted on was arterial, and obtained from the cutaneous capillaries by acupuncture.

Experiment No. 3.—I next tried the effect of keeping the slides in an atmosphere of oxygen, and so marked was the effect apparent on the activity of the embryos and their longevity, increasing this two and a half days beyond that previously

observed, that I was led to attempt, by the following method, whether development could be artificially induced.

Experiment No. 4.—Taking two test-tubes sufficiently wide to admit slides narrowed for the purpose, and charged with To-ah's arterial blood, I connected them by tubes with a pipe leading from the oxygen receiver. Turning on the tap, both test tubes were thus kept constantly filled with oxygen. I then placed them under a sitting hen. One test tube was reserved for occasional examination, a small tap being fitted to the tube by which it was connected with the main gas-pipe, hoping that if any results showed themselves I might, after a suitable interval, be able to get these more matured in the unopened tube. At the first examination, however, I discovered that coagulation had proceeded to an extent incompatible with the existence of the filariæ, at least I thought to this was due the disaster which seemed general. No doubt the continued high temperature under the hen conducted to the speedy formation of clots. In reference to this, it is very necessary in making experiments as to the longevity of the embryos and their solubility in the liquor sanguinis, as I pointed out in my former paper, when referring to these, that both slides and cover-glass should be carefully oiled for about a quarter inch round the sides, before adjustment, when it will be found that this not only prevents dessication, but also seems to arrest coagulation. If unoiled slides are used, evaporation takes place very rapidly, clot forms, and the embryos, however vigorous, are very soon killed.

Received into the stomach of the mosquito, the fluidity of the blood seems to be preserved all the time it is in that organ. The leech, as is well known, retains the blood for several months in its stomach uncoagulated; this is doubtless due, in great part, to the contact with living tissue, affording no scope for liberation of the fibrinogenous ferment. The following experiment would seem to lend force to the supposition that the absence of oxygen is the cause of embryo mortality.

Experiment No. 5.—I took two slides, A and B, and charged them with blood from the finger. They were, of course, carefully oiled, and every precaution taken in their selection to ensure the safety of the embryos.

Slide A. was found to contain five filariæ; round all its sides was carefully painted a solution of Canada-balsam in chloroform, the glass being so held, as the application was made to each edge, that risk of the vapour getting between the glasses was obviated, or reduced to a minimum. When

quite dry, a solution of ordinary sealing-wax in alcohol was painted over in like manner, so as to hermetically seal the included fluid from the outer air. The embryos were immediately examined, to make sure that no injury had been done them by the operation, and they were found to present all the appearance of health and vigour. Not so, however, two hours afterwards, at which time examination showed them all to be feeble, and stretched out; one was apparently dead. *Twelve hours afterwards* not an embryo, or even the remains of one, was to be seen on the slide, though no signs of dessication or coagulation were apparent, the blood-globules rolling about freely on being shaken.

To make sure as to the vigour and general salubrity of the filariæ on *Slide* B, it was not sealed up until twelve hours after preparation, and prior to doing so was carefully examined; the parasites were seen to be vigorous, and presenting the appearance common to healthy specimens. The same process was again carefully gone through, and immediately afterwards the slide was examined to discover the condition of the embryos, of which there were ten. Six hours afterwards only *seven* were visible, all very feeble, and in twelve hours they had all disappeared. Slides simply oiled, but unsealed, prepared at the same time as these, preserved the contained embryos for the usual period. It may be, of course, that in spite of all precautions and apparent success in prevention, the vapour of chloroform got in between the glasses, and brought about the result, *but I do not think this was so.* However, the foregoing are the results of my observations, and of course require further and corroborative investigation by independent observers, before the conclusions I have been led to adopt can be accepted generally.

To sum up:—The following conclusions are those I am inclined to submit as seemingly borne out by what I have attempted to describe.

First.—Accepting that parturition in the parent worm is continuous and exceedingly prolific, then removal of those filariæ which have had their chance of mosquito-withdrawal, but have escaped selection, *is necessary*, in view of the swarm which will inevitably enter the blood, from the lymphatic system, at the next period of its suitability for their reception.

Secondly.—The marked difference between the condition of that fluid when favourable for filarial development, and its state when hostile to their existence, seems due, either to the *presence* of an excessive amount of carbonic acid, *or* the *absence* of a sufficient quantity of free oxygen, inasmuch as the

former necessarily involves the latter, and experiments seem to show—

(*a*). That direct contact of carbonic acid in presence of excess of oxygen is not immediately, at least, injurious, but that—

(*b*) Exclusion of oxygen, either by surrounding the blood with carbonic acid, or placing it in a condition opposed to access of the former gas, brings about the destruction of the parasites; and that,

(*c*) The behaviour of the filarial embryo, both in the human blood and when contained in the mosquito, seems strongly to indicate a desire for this gas, we may assume that, not improbably, its presence is essential to further development, and its withdrawal, or diminution in quantity, conducive to death.

I would therefore suggest the following as the course pursued by the embryo from birth until its absorption by the mosquito. For a period of not less than twelve hours, and probably in no case exceeding twenty-four, the filariæ remain in the lymphatic system; at some time during this period, urged by the favourable conditions in the hæmic circulation, it exhibits for the first time a selective ability, and enters the latter. It will be observed that, supposing twelve hours the least time apportioned for intra-lymphatic existence, and that the filariæ disappear from the blood at 7 A.M., appearing again at 7 P.M., all those born between these hours will be ready for entrance into the blood at various times during the following night; but those born during the night itself would only be fitted for intra-vascular existence at some time during the day.

This infers a longer or shorter delay, according to the hour of birth, and may account for the fact of embryos being occasionally seen in the blood during daylight, as was the case with all Manson's early finds, got as they were during that period. It is possibly, however, more or less the result of accident, due either to what we may call impulsive injudiciousness on the part of a few, or untoward subjection to the irresistible forces present at or near the termination of the thoracic duct, within the scope of which they have been tempted to approach too closely.

As to the facility for postponing their advent shown by those embryos born during the night, we have actual proof of such capability, on the part of the parasite, in those geographical results I have previously described; for taking the case of the man from India, the embryos seen in that country, and those visible in England, being presumably from the

same parent or parents and her parturitive routine, we must suppose remaining unaltered, those of the young which appeared at 7 P.M. in the East must have, at least, been able to defer their *début* for over five hours when the man arrived in London. Applying this same reasoning to the hypothetical Fijian, we arrive at twelve hours as probably the extreme limit of restraint.

I need scarcely point out that the term "postponement" is only used in a figurative sense, implying that the products of certain hours that appeared at 7 P.M. in India would, if keeping to the same arrangements, appear five hours too soon in the blood in London, and of course compliance with the necessities of travel is really only an adjustment in sequence.

Although, as before pointed out, I believe the embryos are wanting in locomotary power at this stage, still there can be no doubt of their ability to resist moderate onward pressure, either by actual adhesion, which, however, I do not think very probable, or, as is more likely, by the opposition of forces, resultant from the contractions and extensions in the body, converging as these do, towards a central point; they are able to control, within certain limits, the duration of their stay in the sluggish lymph current. When the proper time arrives, however, they yield to the flow and their passage to the now attractive blood is brought about.

Note that in the mosquito, where migration has to be effected in a stagnant medium, inherent locomotary powers are required, so at or about the end of another twenty-four hours—observe the coincidence in duration of period—the embryo temporarily becomes possessed of this ability, moving about freely. Arriving at the thorax, and beginning a stage where quiescence is more conformable to its necessities, the parasite seems again to enter on "a sort of chrysalis state", or, in other words, to become passive.*

To briefly summarise the above: being the results of continuous parturition in the parent, the embryos pass a certain term of existence in the fluid on which, when matured, they will depend for sustenance. Proceeding from this in their progress towards development, their next environment is one where the essentials for such are amply provided. The mosquito then steps in, and once more conditions suitable to filarial requirements are afforded; and, lastly, at maturity they find themselves deposited in a position most favourable for transport to their future and permanent habitat. Thus, all the embryonic stages are passed in different media and under various conditions.

* See Manson, *Customs Medical Report*, No. xiv, p. 12.

From the very offset, the young embryo, as it is launched on the first important phase of its career, is made to enter the blood at a point from whence it may most speedily be in contact with that which is so essential to its vitality and growth. Free of the thoracic duct, rapidly floated through subclavian vein and heart, it quickly reaches the lung, where oxygen in abundance refreshes and strengthens it, prior to starting on the circuit from whence insect-delivery becomes possible. Failing to secure this, and returning exhausted, once more the invigorating process is undergone, and so on, until that time arrives when the competition is decided, and room has to be made for the eager throng that in turn must be afforded a chance, nay, are even then preparing for the coming of night with its opportunities. The rejected must go! The choice for survival has been finally made! This is accomplished by no sudden or irregular convulsion, but by the quiet and orderly necessities of another and greater economy. A change takes place, the nurturing host becomes the ruthless destroyer, the equilibrium of nature is preserved, and she pursues the even tenor of her routine: the harmony of the whole conduces to the excellence of its parts.

I now come to discuss the bearing Filaria Sanguinis Hominis has on elephantiasis; but before entering fully on this, a preliminary consideration of the leading characteristics of the disease may facilitate further inquiry.

Sir Joseph Fayrer, both before the Pathological Society in February 1879,* and in a lecture,† states his belief that elephantiasis is a disease peculiar to tropical climates, and distinct from those affections met with elsewhere which present, however, somewhat similar appearances.

Dr. Stephen Mackenzie, on the other hand, thinks that no definite line can be drawn between dermatitis and elephantiasis Arabum, and in fact that it is only a question of degree.

Mr. Jonathan Hutchinson,‡ in a Lecture delivered at the London Hospital, while clearly differentiating this form of enlargement from other morbid conditions in which increase of size forms the most prominent feature, sums up his description with the following graphic statement:—

"*Inflammatory disturbance of nutrition is the starting point.*" The italics are mine. "The tissues are flooded with serum, and owing to their dependent position—scrotum, labium, or leg—this serum has difficulty as to its reabsorption.

* *Lancet* (1879), i, p. 267.
† *Ibid.* (1879), i, p. 433.
‡ *Ibid.* (1876), ii, p. 282.

The cells of the part, already in a state of excitement, feed on it, and irregular modes of growth are the result. You might obtain a somewhat parallel phenomenon if, to any given village, unlimited supplies of beef and beer were freely consigned for gratuitous distribution." From the various general and minute investigations, made by pathologists at different times, the chief morbid changes may be summarised as follows: Epidermis thickened, enlarged papillæ, dermis enormously thickened, and its tissue looking as if infiltrated with a clear fluid; hypertrophy of subcutaneous connective tissue, due to increased cell-proliferation, which latter state Vandyke-Carter* says may extend as far as the periosteum; dilatation of the lymphatics, extending, according to the same authority, as far as the thoracic duct; sweat-gland ducts elongated; blood-vessels numerous and enlarged; nerve connective tissue thickened.†

M. Cornil, in a special and extended examination, particularly mentions finding the lymphatic glands in a state of chronic inflammation.‡ The foregoing appears to be a fair *résumé* of the pathological points on which all observers seem to agree, and which my own investigations, so far as they go, certainly support. Although, as far as I can discover, Sir Joseph Fayrer stands alone in the supposition that the elephantiasis Arabum of tropical or sub-tropical countries differs essentially from that condition which has been described as, and asserted to be by so many observers, the same, though occurring beyond the sphere defined by Sir Joseph Fayrer, still, as coming from this distinguished Indian surgeon with his vast experience, any opinion most deservedly carries great weight, even in the face of testimony from witnesses whose number and exceptionally high standing would otherwise render their evidence incontestable. Referring to some of these, I find Mr. Jonathan Hutchinson, in the lecture previously quoted, enters minutely into the pathology, expressing a strong opinion, and giving illustrative cases.

Further, Mr. Bryant's case, § Mr. Holmes', ‖ Mr. Alcock's, ¶ Mr. Carr-Jackson's, ** Messrs. Cocker and Hill's, †† Dr. Stephen Mackenzie's, ‡‡ Dr. Crocker's, §§ and Mr. Francis Mason's, ‖‖ all of which are distinctly described as elephantiasis Arabum,

* *Lancet* (1873), i, p. 37.
† *Ibid.* (1870), i, p. 268; (1880), i, p. 565.
‡ *Ibid.* (1883), ii, p. 554.
§ *Ibid.* (1886), i, p. 147. *Ibid.* (1874), ii, p. 587.
‖ *Ibid.* (1866), i, p. 147.
¶ *Ibid.* (1866), i, March.
** *Ibid.* (1866), i, p. 396.
†† *Ibid.* (1868), i, p. 378.
‡‡ *Ibid.* (1880), ii, p. 619.
§§ *Ibid.* (1880), ii, p. 619.
‖‖ *Ibid.* (1883), i, 411.

occurring in the United Kihgdom, in subjects who, I gather from the absence of any statement to the contrary in some instances, and direct assertion of the fact in others, were natives of those countries, and always resident there. Again, before the Pathological Society of London,* in a discussion raised on some cases shown by Sir Joseph Fayrer, with special reference to their filarial origin, Dr. Tilbury Fox stated that "several cases have occurred in this country" (United Kingdom), "and that the anatomical changes of the skin in the" (Indian) "cases described agree with those he has himself observed." The foregoing are all cases of elephantiasis of the lower extremity; the following is, however, I imagine a very typical case of elephantiasis scroti, arising and treated in England.

Mr. Bickersteth of Liverpool, who reports the case,† styles it "a large scrotal tumour", laying special stress on the fact of there being a fibro-cartilaginous growth embedded in a mass of hypertrophied skin and sub-cutaneous tissue, attached by a tough fibrous material to the upper part of the scrotum, towards the position of the left external ring. It would appear also, from the history given by the patient, that the growth commenced in the groin, and afterwards slipped down into the scrotal sac. For some time it remained freely movable, the patient being able to slip it up and down from groin to scrotum; gradually, however, becoming fixed in the scrotum, it slowly increased in size, and would seem to have excited the morbid condition with which I am specially concerned at present. At a further stage of this inquiry I shall have occasion to again refer to this case, as one certainly contributing to, if not positively confirmatory of, the theory as to the hypertrophic lesion being a consequence of an excitant which may assume any form, and which under certain and favouring circumstances can induce tissue changes, bringing about a condition not necessarily confined to any one locality or race by anything specific in the ensuing disease itself.

Mr. Bickersteth reports on the mass, after removal, as follows: "The greater part of it consisted of hypertrophied scrotal skin and sub-cutaneous tissue, the latter so infiltrated with serum as to present an almost jelly-like appearance. Embedded in this was the original growth about the size of a thirty-two pound shot, enclosed in a firm capsule, and presenting on section all the appearance of a fibro-cartilaginous tumour; subsequent microscopic examination showed this to be its true structure. From its upper part a firm band

* *Lancet* (1879), i, p. 267. † *Ibid.* (1871), ii, p. 187.

had passed upwards, which was divided in the course of the operation as above described." This speaks for itself as to the pathology, and needs no comment from me. I may mention, however, as a further coincidence, that a tough semi-gelatinous mass is often found extending from the testicles high up in the scrotum to the lowest part of the latter.

Dr. Manson, in the *Customs Medical Report* for Jan.-Mar., 1872, p. 28, when describing his operation for scrotal amputation, specially notices this; and I remember on the only occasion I had the privilege of seeing that gentleman operate on such a case, a band similar to that described by Mr. Bickersteth passed up to a position so suspicious as to cause Dr. Manson to hesitate while we held a brief consultation touching the possibility of its connection with a hernial sac. This structure may be merely the hypertrophied remains of the gubernaculum, thus rendered conspicuous.

Fairlie Clarke, in his *Manual of Surgery*,* mentions having seen an elephantiasis of the scrotum "which, when removed by Mr. Wiblin of Southampton, weighed nearly thirty pounds, also one removed by Sir William Fergusson,† from a bricklayer who had never been out of England, and who had attributed the commencement of his disease to a blow." Then there is Liston's celebrated case; while with reference to elephantiasis of the leg, the same author (F. Clark) quotes Dr. C. J. Richardson's case of a young woman who also had never been out of England. The illustration accentuates this latter as a typical one. No doubt there are several others available to one with greater means of reference at his disposal than I have, but perhaps these may suffice to show that true elephantiasis occurs in temperate climates, though much more rarely than in the tropics, or that the supposition has, at least, a very plausible history to support it.

Again, on more critical examination of the two sides, as represented by Sir Joseph Fayrer on the one hand, and the European authorities on the other, the difference may be found less irreconcilable than at first sight appears, or is at any rate capable of more satisfactory explanation.

Thus, assuming hypertrophy to be a condition set up by the presence of *some* excitant, without reference to the geographical location of the subject, one can see how, supposing in any given area exciting causes were more abundant, and climatic influences favoured the tendency in the tissues to respond to the stimulus in these districts or countries, the number of persons affected would be undoubtedly greater than those residing in places where irritants were less common,

* Third edition, p. 336.

† *Lancet* (1861), ii.

and the tissues less inclined, by hereditary or climatic influences, to resent their presence. Hence, although the disease must be spoken of as endemic in the one, and sporadic in the other part of the world, this qualification is only governed by the prevalence of exciting causes peculiar to a given locality, while the consequences are *pathologically* the same everywhere.

Reflecting for a moment on the manner by which plasma-exchange takes place between blood and the tissues in the normal state, one is reminded of the fact that, strictly speaking, the only difference between this process and that obtaining after a morbid condition has been induced (inflammation), is one of degree, and this would seem to hold, even though we consider the primary nerve impulse leading to blood-determination or congestion, natural lymph-flow or morbid effusion; so these results likewise vary in direct ratio to the activity of the stimuli calling them forth. From the demand made by a part in its healthy functions, through "increased exercise of this, or irritation short of exciting inflammation",* to the extreme disorganisation of nutrition, ending in molecular disintegration and death, we find the same principle in action. Then working back from this last state we see resolution progressing stage by stage, until the normal equilibrium between tissue-want and lymph-supply has been re-established. In other words, it would seem we arrive at a relation between hypertrophy and inflammation, *vis-à-vis* the original stimulus, that is in great part dependent on the amount of vigour or permanence of the latter, affected to some extent by induced or existent regional susceptibility.

Hypertrophy standing, as Sir James Paget shows, on the neutral ground between healthy function and inflammatory action, can, we may suppose, assume proportions bringing it so close to the latter state as to render definition of a distinct boundary line in all portions of the affected region difficult; or, perchance, the state itself induces concomitant changes which may be more properly included under inflammation; and this, if so, would account for certain seeming variations from the typical pathological appearances, common to elephantiasis wherever met.

Admitting, then, that the primary cause of elephantiasis is *some* kind of excitant, one can readily surmise how completely the mature filarial parasite in the tissues might come to fulfil this *rôle*, not, perhaps, at once, or even at all, if taking up a position remote from sensitive surroundings, such as in a large lymphatic, where both presence and functions would be

* Paget.

more or less adapted to its environment. Even if fixed beyond this system, in tissue itself, it may well be that a solitary filaria could be accommodated, and bring forth offspring, without materially affecting the normal equilibrium. If, however, for any reason, the parent worm took on a morbid state, such, for instance, as would be shown by its premature discharge of the embryo, then, by reason of its own altered state, coupled with the abnormal condition of the progeny, it seems possible that the amount of subdued excitement necessary for producing hypertrophic changes may be readily afforded; or if the parasites are numerous—and how easily this may be brought about we can imagine, when remembering that the subject himself is an ever prolific centre of transmission—the peculiar frequency of the disease in these localities, as compared with that prevailing in other places not so qualified, can be accounted for. That hereditary influences have marked effect in favouring the result under discussion is verified by the experience of most observers, but I will only allude at present to that of Mr. Bryant, who quoted to the Medical Society* the case of two lads, sons of a West Indian, but brought up in England, who developed elephantiasis while there. The same gentleman† further stated that he knew of a case in Leicester where the disease had been transmitted through three generations, none of the patients having ever left the town. Race susceptibility is frequently ascribed as another predisposing factor. No doubt people indigenous to certain regions, where external exciting causes are common, acquire the disease, and transmit a proclivity to their posterity while resident in that part, but if large numbers of the same race be transported to countries where these primary causes are rare, after a few generations they do not seem to afford more instances of disease than do the natives of their adopted land. At least, I believe this to be the fact with the African negroes compelled to settle in some parts of the United States of America. Some may, therefore, think hereditary tendencies acting through several generations subjected to continued risk, will more correctly cover what, *primâ facie*, and under certain circumstances, might seem to be characteristic of special peoples.

With reference to the question as to the liability of any form of irritation to set up elephantiasis, I would refer to the following as strongly tending to support such an hypothesis, all being cases where the existence of emboli, or other obstructions to lymph-flow, has not been, and possibly cannot be, suggested as inducing the state referred to. First, Mr.

* *Lancet* (1883), i, p. 411. † *Loc. cit.*

Alcock's case* originated from a dog-bite, the enlargement beginning as soon as the wound had healed. Mr. Carr Jackson's† commenced with abscess in "the lower part of belly". These cases occurred in England. From Bengal,‡ Dr. Hamilton reports a case that commenced from a tulwar cut received below the knee. Even by my theory, this of course may, in that country, have been merely the final stimulus needed to complete the action other causes would eventually have brought about unaided.

Dr. Stephen Mackenzie§ reported a case from Ireland to the Clinical Society of London, which began ten years previously with an injury to the leg, and stated that he had been informed by Mr. Barker that cases of elephantiasis were not so rare in Ireland as in England. Dr. Dowse, at the same meeting, also mentioned a case of his, due to syphilitic infection.

Dr. Heath-Strange‖ showed the Medical Society of London a remarkable case of elephantiasis of the thigh, apparently consequent on vaccination. Mr. Francis Mason, the President of the Society,¶ also alluded to a similar case under his care; and, in still stronger corroboration, Dr. Routh¶ *not only stated that he believed it possible to produce the disease artificially, but mentioned having actually seen this done by an hypodermic injection of amyl nitrite, accidentally administered instead of morphia.*

Mr. Jonathan Hutchinson** states as follows: "In English practice we meet with the two varieties (nævoid and smooth): in most cases of the tuberculated form the elephantiasis takes its origin from some local injury, or local source of inflammation, an ulcer on the leg, an attack of eczema, or, on the genitals, venereal sores may be its starting-point; the smooth form, however, being usually without such cause, and is often set up by a form of inflammation somewhat resembling erysipelas. We have, in both forms of elephantiasis, a very interesting illustration of the results of over-feeding of tissues."

Referring back to Mr. Bickersteth's case, the following quotation from that gentleman's report may be thought pertinent to the question immediately before us. The patient's "statement with regard to the scrotal tumour, of which he was the subject, was that seventeen years previously he noticed there was a small lump about the size of

* *Lancet* (1866), i, March 24.
† *Ibid.* (1866), p. 396.
‡ *Ibid.* (1879), ii, p. 649.
§ *Ibid.* (1880), ii, p. 619.
‖ *Ibid.* (1883), i, p. 411.
¶ *Loc. cit.*
** *Lancet* (1876), ii, p. 282.

a bean in his left groin, situated near the lower end of Poupart's ligament." One day, after retching violently, and "while straining, in the act of vomiting, he felt this lump slip down into the scrotum. For quite twelve months after this occurrence the lump remained movable, so that he could slip it up into the groin at pleasure. Gradually, however, it became fixed in the scrotum, and slowly increased in size, dragging down with it the left testicle to a lower level than the right one. For the first seven years or so the tumour slowly but steadily grew, till it was about the size of two fists, not causing any pain, but after that time it grew more rapidly and soon doubled its size. As the tumour grew, so the scrotal integuments and structures beneath all took on a hypertrophic action, and also increased in bulk. Finally, the tumour and its hypertrophic covering attained an enormous size."

If the above be accepted as conclusive of the proposition, then we can see how the Filaria Sanguinis Hominis might very frequently, through some untoward disturbance to its possibly otherwise harmless existence, become a fruitful source of elephantiasis; while at the same time, though to lesser extent, because of their comparative rarity, other causes, perfectly different in themselves, might, in places remote from those where this parasite is found, produce exactly the same pathological results. No doubt the filaria is a very potent and, indeed, somewhat general source of mischief, for Dr. Bancroft told the Medical Society of London* that "he could give a list of thirty different diseases connected with 'filariæ', while he also added that, having examined many cases of elephantiasis (presumably, in some instances at least, *post mortem*), no filariæ were to be found."

Sir Joseph Fayrer, therefore, may be quite justified in speaking of the endemic elephantiasis of India, set up by purely local excitants, and fostered by climatic influence and predisposition, as something which, *in relation to primary cause and intensity of effect*, differs *in these respects* from the disease occurring elsewhere, and may be amenable to treatment (*e.g.*, "removal from place to place", etc.) which in cases where the causes are less persistent, or in their nature not requiring such measures, would prove unnecessary. On the other hand, those who accept elephantiasis, *per se*, as *pathologically* the same *everywhere*, might also be held accurate to the full extent of their assertion.

That Filariæ Sanguinis Hominis do take a very preponderating, if not sole, share in the causation of elephantiasis and

* *Lancet* (1873), i, p. 465.

allied diseases in many countries, I scarcely think can be denied; nay, I am tempted to go further, and submit that in places where other sources are now suspected, or accepted, examination may yet prove Filaria Sanguinis Hominis the chief offender.

Even in districts like Amoy, however, where one in ten of the people are infested, I would not be surprised to hear of an occasional case of elephantiasis where the closest examination, both *ante* and *post mortem*, not only failed to discover the parasite, but, on the contrary, favoured the supposition which the previous history might set up, as to another and quite different commencement.

I would suggest that, though in this hypothetical instance the disease be genuine elephantiasis in all its aspects, such need not in the least detract from the fact that in the vast majority of local cases the cause so justly suggested and ably supported by Manson as acting in that district, is one which, in the interests of millions, practically demands the undivided attention of medical men resident in the east.

Although I have generally confined myself to the use of the term elephantiasis, if the views I suggest of that disease be deemed satisfactory, lymph discharge, whether from the surface of the skin, such as in "milk scrotum", or into the urinary track, "chyluria", would seem to be accidents of the main disease, which, when they occur, by providing an outlet for the superfluous lymph, deprive the tissue elements of the opportunity for undue voracity, and thus arrest, or modify, the hypertrophic changes that we observe in parts deficient of such relief. This may also account for the comparative rarity with which elephantiasis is associated with lymph-discharging phenomena.

In the cases where the two are combined it is, perhaps, not too much to assume that the external flow has been established at some period subsequent to the hypertrophy, or, perhaps, in the course of its progress; when we may expect, and as a fact do find, the growth, if not actually checked, so considerably modified as to lead to a reasonable suspicion of intimate relations between the two manifestations.

Mr. Carr Jackson, when referring to his case, states as follows: "The tortuous lymphatics occasionally burst naturally, and discharged freely. The boy states that he has collected pints of it. The limb has evidently decreased in size since the lymphatics came to the surface and have disgorged their contents from time to time." With chyluria, no doubt the ready exit which is offered for the discharge,

consequent on the frailty of the comparatively unprotected and tender vessels in that locality, affords an early means of arresting a condition which if set up in those regions might undoubtedly be fraught with much graver consequences than immediately follows hypertrophy of less vitally important parts. It would almost seem, therefore, as though these phases of the disease were natural efforts to avert results that would ensue if all the lymph exuded was available for tissue consumption; and while from it we may possibly foreshadow the beneficial effects that so many assert have followed the artificial attempts to limit plasma supply to already overfed tissues,* by more closely investigating these occurrences some useful hints may be obtained for perfecting a curative measure, which though correct in principle, undoubtedly lacks perfection of detail and constancy of effect. In strong confirmation of the supposition that lymph-scrotum and chyluria are only phases of one disease, I would refer to Dr. Manson's work *On Filaria Sanguinis Hominis*,† where he most exhaustively, and, to my mind at least, convincingly proves, by clinical evidence and characteristically concise reasoning, the common origin of these diseases with elephantiasis. This allusion to that able observer and indefatigable worker, naturally leads me to the consideration of his theories as to the part Filaria Sanguinis Hominis plays in the causation of elephantiasis and allied diseases.

Briefly, Dr. Manson suggests that the primary cause of the disease is obstruction in the lymphatic glands, plugged as they become by premature discharge of immature filariæ confined in the unstretched chorion, thus forming, together with the enclosed organism, an ovate body, considerably larger than the full-term embryo, five times larger than a lymph-corpuscle. Dr. Manson believes that these gradually plug the afferent vessels in the main lymphatic glands, obstructing lymph flow, and leading to the lesions indicated. There can be no doubt but that this hypothesis has been put forth with all the ability and attractiveness of which Dr. Manson is so well capable, and it is only after long consideration of the conditions suggested by that gentleman, together with known anatomical and pathological facts, that I have at last reluctantly to admit that his arguments do not seem to me so convincing on this point as on many others. If Dr. Manson's view be accepted, we must assume that elephantiasis and its allies are secondary to lymphatic obstruction, *i.e.*, follow a *mechanical*, in contradistinction to a *physiological*, origin; but this would seem to be more correctly applicable to a

* Arterial ligature. † H. K. Lewis (London, 1883), chap. v.

less chronic condition, and although in view of the valves in the vessels, it does not seem easy to agree with Dr. Manson's theory as to "regurgitation" being the cause of lymph diffusion in elephantiasis, one could see how, if from any cause the lymphatics were obstructed in any portion of their continuity, the pressure from behind would rapidly fill and distend the intervalvular portions of the tube, giving to the whole that cord-like, knotted feeling pathognomonic of œdema following interference with lymph-flow. Now this condition of the vessels is notoriously absent in elephantiasis; on the contrary all observers seem to agree that although the lymphatic coats are hypertrophied the lumen of the vessel is uniformly enlarged, indeed Dr. Manson himself pointedly calls attention to this fact in more than one instance. Of course, if, as a consequence of the continued and increasing incitement, the vessels are unable to remove the plasma as fast as it is exuded, *accumulation* takes place in the affected part, and the sodden state described by pathologists is produced. But this would then be due to the proportional inequality between the outpour of lymph and its on-flow, though this latter may be even hastened, and actually passing through tubes capable of conveying abnormally large quantites. This would also account for the tissue-saturation being localised in the neighbouring parts, in some instances in apparent opposition to the laws of gravitation.

Mr. J. Hutchinson, in his lecture before quoted, most distinctly defines and points out the difference between œdema, primarily due to lymphatic obstruction, and that concomitant with tissue overgrowth. For instance, when speaking of the former, he says:* "In lymphatic cases the disease is almost always non-symmetrical; we may note also, as a curious fact, that very commonly no enlargement of the lymphatic glands occurs. I should be inclined to suspect this cause (obstruction of lymph) in any case in which the œdema was strictly local and abruptly limited, there being no evidence of disease of the veins or mechanical pressure. I should consider my diagnosis confirmed if the œdema cleared off without leaving any dilatation of superficial veins; and during the progress of the case I should repeatedly and carefully examine the limb, in order to ascertain if any little lines like whipcord could be felt under it. Indeed, it is not unlikely that primary disease of the lymphatics is extremely rare, and that almost always it is secondary to inflammation of the skin and subcutaneous tissues."

* *Lancet* (1876), ii, p. 282.

Then passing to his seventh group, elephantiasis, he says: "There is also overgrowth; and here we establish the line of demarcation between elephantiasis and all other varieties of persistent œdema; prove that the tissues have become hypertrophied, that they are not sodden with serum, but they are overgrown, and you prove the right of the malady to the title of elephantoid. Size ought never to be made a basis for classifications, and between the condition of chronic thickening of skin with solid œdema and papillary growth, which are not at all uncommon in our out-patient rooms, and the most hideous example of Barbadoes leg which you could find in the West Indian Islands, there is no distinction except that of degree, the pathological process is precisely the same in both."

What chiefly concerns me at present in the above quotation is the support given by the distinguished lecturer to the view that primary affections of the lymphatics are extremely rare, and that when they do occur a condition is set up, not only failing to agree with, but distinctly differing from, that which obtains in elephantiasis; assuming this point unsettled, however, there seem to me to be other and very important difficulties in the way of Dr. Manson's hypothesis, if, with my friend, we imagine the parent worm situated, say, somewhere in the thigh, and that she miscarries, the ova being conveyed to the afferent vessels in the capsule of the inguinal gland. Here suppose the ova to be distributed over the capillary plexus, and *seriatim* plugging them, then, as far as that gland is concerned, its functions would cease, and the process would be carried on until all its fellows were similarly blocked.

We thus arrive at that condition of lymph stasis which Dr. Manson assumes must be produced before the hypertrophy commences; but at the same time, if we admit this, we must also grant that the circulation of lymph over the whole of the lower extremity is now stopped, and the continuity of an important, if not thoroughly understood, nutritive circuit has been suddenly broken.

Thus one might expect the functions of the whole economy to be seriously affected, supposing lymph return essential to the completeness of the general nutritive system, and most certainly would this appear to hold good of the region, so suddenly debarred from eventually taking that donative part, which, to great extent, modifies the effect of deficit in the general store, consequent on its primary demands. Hence, though an obstruction in the lymph vessels, strictly circumscribed in its effects, may for a time be borne, so soon as this lesion involved a large area, and comprehended a duration approaching the chronic, changes more marked than mere

local hypertrophy would be induced, and the system, as a whole, speedily exhibit consequences very different from those present in even the most advanced cases of elephantiasis. I do not understand Dr. Manson to mean, nor does it seem consistent with his theory to suppose, that anything short of complete and nearly simultaneous occlusion takes place prior to the hypertrophic manifestation.

If it be suggested that, after the occlusion of the glands of one side, lymph return can still go on by the anastomoses with the lymphatics of the other, it must be remembered that the ova could also pass in the same way, and plugging of the glands on both sides would eventually occur. In support of this I need only quote Dr. Manson's statement from *Medical Reports*, xxiii, p. 14, where he writes as follows:—"Anastomoses for a time will aid the passage of lymph, but the anastomosing vessels *will carry the embolic ova as well as the lymph.* The corresponding glands will then, in their turn, be invaded, and so on until the entire lymphatic system connected, directly or *indirectly*, with the vessel in which the parent worm is lodged becomes obstructed." The italics are mine. In fact, on the completeness of this stoppage, on the affected side at least, I gather Dr. Manson holds, the inception of the disease depends. If this be the case, how comes it that the affection is not generally distributed over the whole of the part, or parts, on the distal side of the glands. Thus, if a scrotum is elephantiased, in consequence of the inguinal glands being obstructed, why are the limbs also not *always* affected? Or, if the legs, as is generally the case, be the seat of disease, how comes it that very often the thigh is not only unaffected, but little or no signs of interference with the normal nutritive changes of that part are apparent? Venous absorption would scarcely account for this.

Dr. Manson, in his work previously quoted,* writes as follows concerning "Case 20":—"The integuments of the left thigh, over its inner, anterior, and posterior surfaces, are distinctly elephantiased, from the knee to a point about two-thirds up the thigh; the rest of the limb appears to be quite normal, but in the situation mentioned the skin is darker than that on the corresponding part of the other thigh." And again:†—"It may be objected that the affection of the skin of the thigh in this case was not elephantiasis. If it was not this, it is certainly a wonderful coincidence that his mother, who lived in the same house with him, exposed to the same chances of filarial infection, should develop true elephantiasis of the leg."

* Page 115. † Page 116.

Not to multiply cases of partial elephantiasis, I will only refer to Mr. Carr Jackson's, also affecting the thigh, leaving the leg apparently sound; and here I might remark on the bearing such instances would have on arguments tending to support the hypothesis of lymph gravitation being the cause of overgrowth in the leg. It would also seem improbable that the general manifestation of disease was due to a gradually progressive occlusion of the glands, for supposing the ova capable of permanently plugging the capillaries, in such constant and practically unlimited numbers as are the former, the process would obviously be continued so long as the supply lasted, and until all the glands were stopped up. How does the theory of occlusion agree with the structure and arrangement of the lymphatic system? We must remember that although the main vessels divide up into numerous smaller ones in the capsule of the gland, the central part of this organ I scarcely think can be described as "solid", in the sense I understand Dr. Manson to use the term. Without doubt the cortical follicles and medullary cylinders consist of, comparatively speaking, dense adenoid pulp, round and outside of which, in the sinuses, the lymph filters on its way towards the efferent vessels, and it is here that the reticulum catches and "detains pigment or other-particles", for disposal by the large amœboid lymph-corpuscles wandering about in the lymph-paths, devouring or disintegrating such extraneous matter; but I do not see how the arrest of these could there interfere with the free circulation past them. At any rate, if the gland be plugged on its distal side, no lymph, properly so-called, ought to be found in the organ itself. It might be suggested that possibly the exploratory needle did not penetrate to the interior of the gland, getting no further than the "varicose" capillaries in the capsule. But if this were so, disintegrated filarial envelopes, embryos, and other *débris* of like nature could scarcely be looked for, seeing these are changes proper to the gland, and not capable of being produced in the parts external to it. Dr. Manson, and other observers, repeatedly speak of withdrawing a quantity of "milky lymph" from the enlarged glands; while Dr. Manson quotes case after case where he found "embryo filariæ in all stages of vigour", and "numbers of threads", which he himself suggests are the remains of the "collapsed sheath of the embryo, the body of which had disappeared by absorption or disintegration".* As to the presence of filariæ in enlarged glands, when none appear in the blood, I can personally testify. How these filariæ get into the gland, if the passages

* *Op. cit.*, p. 104.

thereto are all blocked, and not only in solitary or accidental instances, but in great numbers, seems a very formidable difficulty.

If what I have previously submitted is correct as to the solvent effect of lymph, or liquor sanguinis, on the debilitated embryo, then it would seem that, presuming the ovum temporarily arrested in a capillary, before long the surrounding fluid, aided by the back pressure, and the motion of the contained embryo, not perhaps in every instance entirely disintegrating, would, at any rate, so modify the shape of, and it may be soften, the embolus, as to allow its passage, at the same time injuring some of the embryos to the extent of causing their immediate death, and subsequent solution in the gland; while others more hardy, having stretched their envelopes, pass on, in a condition less morbid, and so account for the varying stages of vitality exhibited by the filariæ found in fluid taken from enlarged glands. Besides the constriction (if any) on the outside of the ovum (which is doubtless similar to that which enables, or aids, the normal embryo to elongate its sheath prior to leaving the maternal canal) would most probably tend here also to favour a like condition, and thus facilitate a progress only temporarily interfered with by the globate form of the body as first presented. From injury, or immaturity, the embryo eventually perishes in the gland, and this may account for its non-appearance in the blood in advanced cases of elephantiasis.

It also seems reasonable to assume that with increased viciousness of habit, which constant miscarriage sets up, the discharge takes place at progressively early stages of embryonic life, and the offspring are less and less capable of prolonged existence or resistance to intra-glandular influences. Temporary inability of the parent to discharge young with the vitality necessary for withstanding the glandular influences, may also account for the occasional absence of embryos in the blood, their reappearance being due to the recovery of the affected worm, or it may be, of course, to the arrival of a new and healthy parent.

Again, granting that the ova could plug an ordinary afferent capillary, it is fair to assume this must cease as soon as these tubes are dilated to an extent even proportioned to that observed in the larger vessels. Vandyke Carter* tells us that this dilatation of the lymphatics "extends as far as the thoracic duct". Mons. Cornil,† though he found the glands in a state of "chronic inflammation", does not mention, or hint at embolism, or "varicosity" of the capsular vessels, a condition

* *Lancet* (1873), i, p. 37. † *Ibid.* (1883), p. 554.

one would surely have noticed when making the minute examination that eminent pathologist describes.

Apart, however, from the fact of embryonic presence in the glands seeming to point to patency of its approaches, ought we not rather to expect a state of atrophy in an organ the functions and utility of which have been so completely interfered with; but then, could such a condition have escaped the notice of observers like Dr. Vandyke Carter, Mons. Cornil, and others who have had opportunities of making minute *post mortem* examinations? Or would such be consistent with the chronic glandular enlargement observed during life, and which Mons. Cornil and others have shown to be due to inflammation of the connective tissue in this structure.

The foregoing are some of the chief difficulties arising in my mind when considering Dr. Manson's pathological views; I trust, however, that whilst attempting to define them, I may not have appeared either dogmatic or hypercritical. Well aware of the immense difference there is between the labours of one who has to construct, from most limited data, and the comparatively facile task devolving on his critic, I am also fully conscious that in stating what follows, the position assumed is equally liable to merit adverse criticism. I know also that whatever theories I advance in my turn, are put forward with all the diffidence which the inadequate means at command necessitate, and can merely, in the absence of far more extended investigation, be taken as somewhat crude ideas, fortunate, if only useful in stimulating the attention of more able and favoured observers.

Assuming the primary cause of tissue overgrowth to be *physiological*, rather than *mechanical* in its origin—that is to say, that the excess of lymph effusion is itself an intermediary consequence between the primary excitant and its most marked result—I would submit that whatever was capable of setting up increased plasmic flow, as distinguished from accumulation due to obstruction (being short of inflammatory effusion), whether it be the Filaria Sanguinis Hominis, a wound, an ulcer, or an eruption, provides the means for tempting cell voracity, and so produces all the manifestations at present under discussion; whereas the condition resulting from mere lymph stasis, due to a cause entailing suspension of an important circulatory function, would induce a morbid state, acute in its nature, and not compatible with the sub-normal condition favouring hypertrophic changes. Let me suppose the filarial parent located in any given part; so long as it remained the sole invader of those regions, it seems quite possible that the natural powers of adjustment might obviate any

material disturbance of nutritive equilibrium. Though a demand for slightly excessive supply might be complied with, and no great disarrangement between the outcome of lymph and its natural removal occur, still, should this requisition be made too often, then it might probably come about that the response assumed proportions beyond the capability of even the additional parasitic consumers to cope with; and in the first instance, if only to render the task of the absorbents less arduous, the tissue elements, prompted to undue participation, become, as a consequence of their indulgence, still greater stimuli, leading to freer lymph-flow, and so on. Greater demand is followed by hyper-proportional supply, until we arrive at a condition where, progress as it will, the plasma-contribution always exceeds the power, either of the greedy overgrown tissues to overtake, or the congested lymphatics to carry off. No doubt the filaria takes part in the extra voracity and its consequences, a morbid condition is set up in the worm, she miscarries, a state by the way in itself possibly capable of intensifying surrounding excitability, and then we have that step beyond mere excitation capable of inducing increased functional activity, as pointed out by Sir James Paget, and something more, allied, if not precisely similar, to the inflammatory condition, is set up. At first this may go no further than those febrile manifestations (lymphatic fever) exhibited so generally in elephantiased patients. The embryo filariæ from the affected worm or worms, discharged more and more prematurely, gradually fail in the vitality necessary to carry them beyond the glands, and at last cease to appear in the blood. In a word, I would suggest that after setting the morbid process going, the parasite, beyond possibly tending to continue the excitation, ceases to take any direct part in the action itself, nay, she may herself succumb to its vigour, forming the centre of a defined inflammatory area, and the remains finally find exit, when the resulting abscess bursts or is incised.* Should the host be in a position liable to continued parasitic invasion, of course, with the advent of each worm, the tendency to set up excitation is greater; and when at last the limits are reached, and the filarial irritation, whether by reason of numbers, or morbid changes in one or more of the invaders, becomes greater than is compatible with a perfectly normal condition of nutrition, consequences are induced, and go on until the disease under notice is in full progress. Limited only by the power in the exciting cause, or causes, to extend their effects, we might thus account for the varying position and extent of the manifestations so

* See Manson, *op. cit.*, p. 127, Case 23.

often observed; though, if on the other hand, we attribute their origin to something necessarily and indissolubly affecting a *whole* region, the frequent restriction of its effects to a portion only would scarcely admit of such simple explanation.

I think the case of the gig-man To-Ah may usefully be quoted here, in illustration of my supposition. This is the same man who formed the subject of my observations and experiments described in the *Customs Medical Reports* for half-year ending March 1881, where he gave the following history: "In October 1879, he was 29 years old, and a native of Amoy, where he resided until he was 21. From the time he was about 14 or 16, he has suffered at various periods from fever and ague. At about the age of 18 or 20 he first noticed swellings in his groin, which, however, have increased but little; in fact, he thinks they show a tendency to lessen in size. He suffers during the hot season from sharp attacks of 'fever and ague', otherwise he is in good health, well nourished, and generally fit for his work. He was not aware he had filariæ in his blood, and does not think much of the fact, though he watched the embryos under the microscope with much interest. He has visited Amoy twice since he first came to Formosa, but as his friends and relations have all died off he thinks of permanently settling here. He does not suffer from any inconvenience whatever when pulling, even long distances, in the gig, nor does he find that he is unfit for considerable exertion of a pedestrian kind, and often accompanies his master shooting, carrying a tolerable weight all the time; is quite willing to allow me to make the experiments explained to him, and will be glad if he can be cured of his tendency to 'ague' altogether, as 'then he would be quite well'. There is nothing abnormal about his scrotum or legs, and in every way he appears an athletic, well-developed man."

At the time I wrote, enlargement of two right inguinal glands was observed; but he told me then, and I afterwards found it to be the case, that other glands occasionally swelled. This generally happened just before and during his attacks of "lymphatic fever". Gradually, however, he has lost all tendency to these attacks, having had no fever of any sort for the last three years, and the glandular enlargements have quite disappeared. He has, I regret to say, become an opium smoker, but to this indulgence he strenuously attributes the absence of lymph-fever. Should this latter be a manifestation of inflammatory disturbance in the way I have ventured to suggest, it is possible the narcotic might so act, in the

absence of increased, or increasing excitants, to keep down a tendency which residence in a locality where fresh incentives were being constantly received was wont to set up. There can be no doubt that, though still vigorous, the average number of filariæ found in his blood at any given time is less than it was six years ago. His general health keeps good, and he works hard now as a mason's assistant, having exchanged this calling for that followed when last I wrote. He has no signs anywhere of tissue overgrowth, and I think has good reason to hope that he is getting over the liability to this lesion. I would suggest that being removed from the district where the filariæ are abundant, and constantly being propagated, to one where the cycle of genesis is abruptly interfered with, as seems to be the case in Formosa, he is freed from the consequences of increased invasion. That the worm, or worms, which had located themselves before he left the mainland, are, happily, either situated in parts indifferent to their presence, or else, if not, then those parasites not so placed, did duly commence their irritative action, as his former febrile seizures seem to show; and had he remained in a district where the number of parents could eventually have been added to, some of these fresh arrivals would in like manner have themselves taken on a morbid state, and so directly assisted in intensifying the irritative action thus set up in the parts, or by further debilitating the original offender, indirectly contribute to this result. No such additional incitement being available, the affected worm or worms, favoured by the comparative quietude, recover their health and cease to be undue incentives to hyperplasmic exudation. Again, it may be that the mere presence of parasites in numbers is all that is required to start abnormal action, and unless plurality is possible, man can, within these limits, act as hospitable host, without discomfort to himself or detriment to his guest. After a time we must assume the filaria dies and is carried away. Should her decease ensue in the course of nature, and not be brought about by any violently stimulative process, we can assume that the remains, like much other apparently irritative matter, may be quickly and unostentatiously removed by the absorbents. If these surmises be correct, they would tend to account for the fact that removal from place to place, and especially out of the district or area where filarially induced elephantiasis is prevalent—so strongly insisted on by Sir J. Fayrer—is likely to be beneficial; while, as helping to control both tissue-greed, and its effects, that which tends to limit plasma-supply must also be adopted in further treatment of the case.

If I might venture on suggestions for treatment, and possible cure of elephantiasis, based on the foregoing hypothesis, I would submit that the *first* and *most essential step should be one towards securing immunity from further infection.* Unfortunately, in those countries where these diseases abound the people are so situated that in the majority of instances, if the carrying out of this desideratum was dependent on removal from one place to another, the advice would be apt to coincide with the proverbial "beef and wine" prescription for the starving pauper. Happily, as far as filariæ are concerned, we have other means for debarring their access, namely, by filtering and boiling all water drank by the patient. With reference to the cure, or amelioration of mischief already set up, looking at the vast amount of testimony there is in favour of controlling blood-supply, and as far as I am at present concerned, its seeming concurrence with the theories now submitted, it would appear that temporary, or permanent obstruction of some main source, is the primary remedy suggesting itself.

Notwithstanding the great consensus of opinion favourable to deligation of arteries, and the apparently excellent results I have myself seen follow such treatment in the early part of my medical career, when placed in a position affording great opportunities for seeing elephantiasis, I am bound to admit that both theoretically, as well as practically, I believe all that is required can be gained by limited pressure, applied occasionally to the main vessel, and more permanently to the affected part. In this hospital, though elephantiasis as a severe and chronic condition, and arising from Filariæ Sanguinis Hominis, is unknown, still a condition pathologically the same, following or concomitant with ulcers or wounds, is sometimes seen, and I have always found Martin's rubber bandages, together with other local and constitutional remedies, effective. With elephantiasis, due to Filaria Sanguinis Hominis, in a district where the parasite flourishes, I can readily imagine such treatment would be at best only temporary, *if not combined with means for preventing further infection,* and modifying existent irritation; but if so associated, then with all diffidence I cannot help thinking occasional pressure on, say, the femoral, either digital, or by tourniquet, together with elastic bandaging, and suitable internal remedies, are likely to bring about most satisfactory results. The *non-combination* of the prophylactic and curative in the East, may account for the want of that success which seems so invariably to have followed even the heroic treatment by arterial ligature in Europe. I cannot help repeating, however, that,

even in the West, it may be open to discussion, whether less extreme measures for carrying out the same principles would not succeed equally well.

One is inclined to hesitate before entirely throwing over the theory on which the procedure is based, because an apparently too severe form of practical application has, hitherto, been adopted, or because it has failed to show sufficiently satisfactory results in cases *where circumstances, outside the procedure itself,* have been antagonistic to a fair test of its efficiency.

I am well aware that what is now suggested is neither new nor original, especially if one takes the recommendations *singly;* but I submit that should the etiology of elephantiasis be somewhat similar to that I have attempted to sketch, then firmer hopes of success, *based on a strict and simultaneous conjunction* of the protective and remedial methods, may be looked for, even in places where the results hitherto have been apparently opposed to western suggestions and accomplishments. It is to be hoped therefore that I may not be thought too sanguine in saying that while agreeing with Dr. Manson, and, indeed, arriving—though by different reasoning—at the same indications for treatment, as those he more than hints at, I own I do not quite share his despondency as to the futility of looking for permanent benefit in many cases of even advanced elephantiasis of the extremities. With advanced elephantiasis scroti, even supposing we could readily apply means for regulating blood supply, little or nothing is to be gained by rejecting the usual treatment. Fayrer and Manson have taught us with what ease and safety amputation may be accomplished; and on grounds of convenience, and comfort, the operation undoubtedly offers the highest attraction. To prevent recurrence, prophylactic measures would certainly be required, and if the flaps showed a tendency to take on diseased action, then it is possible that resort to pressure, etc., might overcome liability to relapse, and complete the cure.

I may mention, in conclusion, that Dr. Routh's statement and experience have so struck me that I have determined on trying a further set of experiments with monkeys to see whether, by injecting nitrite of amyl, I could induce artificially a similar result to that Dr. Routh mentioned having seen follow its accidental insertion under the skin. I am the more tempted to do this as there appears something very analogous, in the action of the drug on the vascular system, to that brought about by the Filaria Sanguinis Hominis, or other excitants, when playing a similar part.

The results from both are alike: vascular congestion and consequent plasma-exudation. The relationship is as striking as it is interesting, and if Dr. Routh's experience is found to be in accord with further investigation, it seems to me, that the evidence in favour of the theory I have ventured to suggest as to the etiology of elephantiasis, and its allies, will have been even more effectively strengthened than by Dr. Routh's single observation, important and valuable as this is.

DIPHTHERIA IN CONNECTION WITH DAMP AND MOULD-FUNGI.

By MICHAEL WAISTELL TAYLOR, M.D. Edin., F.S.A.

(*Read: April 12th*, 1887.)

In a paper brought before the Border Counties Branch of the British Medical Association, and published in their *Journal*, July 2nd, 1881, entitled "The Fungoid Origin of Diphtheria", I gave in full detail the history of a series of cases in one family, in which leakage into the walls of the bedroom, and the subsequent growth of various fungi, were immediate antecedent circumstances to the outbreak of diphtheria. I, in that paper, ventured the deduction, that the growth of common moulds, penicillium, aspergillus, and other fungi, might under certain circumstances become infective sources of diphtheria.

On the present occasion, I will adduce the whole of the observations which constitute the data from which I now seek to verify that conclusion; and the evidence will consist of (1) a certain number of facts derived from my own personal clinical experience, and (2) such observations as have been made by others, or which may have been acquired from other sources, as appear confirmatory or corroborative of the principle at issue.

The problem which still stands to be solved is, What is the origin, or final cause of Diphtheria, or, in point of fact, the disease itself? In approaching the question on this occasion, it will be perceived, in the first place, that we have a certain idea to work out, derived from an interpretation of a number of observed phenomena; or, in other words, a Hypothesis—which regarded primarily, and in the abstract, may be true or false. In accordance with logical method, this is perhaps the best place to set forth the Proposition, which, therefore, in its simplest form, may be enunciated thus:—

THAT DIPHTHERIA MAY ORIGINATE IN SOME COMMON MOULD-FUNGI, GROWING UNDER CERTAIN CONDITIONS.

Now, in the first place, there are some general results derived from statistical references, and the death rates, relating to the distribution of diphtheria, bearing to some

extent on the question of its fungoid origin, which may be considered for a moment. For instance, the special preponderance of diphtheria in rural, as compared with urban districts; the greater prevalence of the disease in low-lying isolated places, and under conditions of wet, retentive soils, as against dry and pervious formations*; of the presence of dampness in the surroundings and walls of houses. Then, again, the numerous instances found in reports of Medical Officers of Health, and of others, of the incidence of diphtheria in the neighbourhood of manure heaps and dung hills,† and of night soil spread on land in country places‡; and in towns the predominant liableness to invasions of the disease of public mews and yards in which stable manure is kept, all point to conditions involving the growth and propagation of mould-spores.

I will first proceed to cite the evidence which has, since my former paper, been forthcoming from other observers, of the concomitance of diphtheria with the presence of growths of mould in dwelling houses, or their surroundings; or with water-supply and milk, which had been exposed to, and might have become the culture media for, the spores of mucors or moulds. Some of these facts have been gathered from cases published in medical journals, and in Public Health Reports to local authorities; some have been communicated to me by medical friends, or derived from personal inquiries.

Case 1.—Mr. H. Nelson Hardy has related§ the particulars of a series of cases which occurred at various times during the year 1885, in an old dwelling-house occupied as one of the Dulwich police-stations. There were seven cases of diphtheria, amongst members of the police force and others, who were successively inmates of the premises, in little more than twelve months. The unsanitary conditions were a state of wetness in the walls of the lower rooms, and a constant damp and mouldiness affecting the floor, the furniture, and the floor coverings, which had persistently endured during that period. The author has recorded the facts expressly to show "that there are good grounds for the belief that there is some special connection between damp houses and the development of diphtheria." But might it not be possible that along

* Dr. C. Kelly, *Relation of Soil to Diphtheria.* Annual Meeting Brit. Med. Assoc., Brighton, Aug. 11, 1886, and Discussion thereon.

† Dr. Highet, Troon, *Med. Times and Gaz.*, 1874. Three cases of Diphtheria traced to a Dunghill within 100 yards.

‡ Dr. Thursfield, "Connection of Diphtheria with Local Unsanitary Conditions" (*Brit. Med. Journ.*, 1873).

§ H. Nelson Hardy, "Damp and Diphtheria" (*Brit. Med. Journ.*, March 13, 1886).

with damp, the presence of the mould may have been implicated in conducing to the results exhibited in this instance?

Case 2.—Mr. Paget, Medical Officer of Health for Westmorland,* refers to a very curious outbreak of diphtheria, in an isolated farmhouse in Reagill in his district, in which eight out of nine inmates were affected within the space of fourteen days in the beginning of July 1885. The local conditions were these—the old farmhouse had been allowed to go out of repair, it was damp throughout from attics to kitchen, and white, green, and grey moulds were found respectively on rotten wood-work, stone, and plaster. On examination ordinary penicillium was seen; and under the microscope a large number of micrococci, and one or two dumb-bell-shaped bacilli. Repairs of the premises were undertaken, entailing the exposure of all this rotten material, whilst the people were in the house; and within six days the first cases of diphtheria appeared. Mr. Paget remarks, "Whether or not the existence and then the exposure of certain moulds can be included with the dampness of the house and the warmth of summer, as a direct excitant, is a question difficult to answer in the affirmative; but, under any circumstances, their exposure is remarkably associated in point of time with the incidence of the disease. In any consideration, moreover, of their possible connection with the outbreak, it is to be noted, that the family had lived continuously in the damp house for fully eight weeks, without the disease showing any sign of its approach, and that only when internal repairs—and thereby exposure of the moulds—were begun did the sickness make its appearance." On the face of the report, there appears to me to be here strong presumptive evidence of the influence of moulds.

Case 3.—I recently visited, for the purpose of inquiry, a house in the village of Lowther, Westmorland, in which the following occurrence had taken place. The family consisted of father and mother, a daughter aged nineteen, and a son aged fifteen. The house was probably over a hundred and fifty years old; it consisted of two stories; a straight passage led from the front door, with a sitting-room on each side; three bedrooms above in front, and kitchen and other apartments at back of the house. The boy occupied the small bedroom above the passage. In the month of June 1886, this boy was ill, feverish, with a sore throat, which was described as being very red inside; he had no medical attendance, but he was at home for over a week from school in consequence. The father himself, also, about this time had inflammatory sore throat, to which he had been subject previously. On

* *Medical Officer's Annual Report for Westmorland*, 1885.

August 23rd, the eldest daughter became ill with painful sore throat, which was reported to be diphtheritic, which indeed was confirmed to me by my friend Dr. Robertson of Penrith, who attended her. The family had come into the house three years before, after it had been shut up and vacant for some time. They had noticed that there was always a bad fusty smell in coming in at the front door, which continued to grow worse. In the month of July, they observed along the course of the skirting boards of the passage and sitting-room, along the chinks of the door jambs, and out of the joints of the flooring, there protruded a yellow, spongy fungus. Soon after the daughter's illness they took down the door frames, and the partition between the passage and sitting-room, and lifted the flooring boards. It was found that the fungus with its great lateral expansions of ochre coloured pileus, described as similar to that growing on dead trees, adhered in great cakes three or four inches thick to the timber of the flooring, to the woodwork of the doorway, smothered the lath and plaster partition, which was penetrated also with fine cobweb threads on which stood dew-drops of moisture. These fungi were probably a *Polyporus*, and *Merulius lacrymans* (dry-rot), which ravaged the old structure in consequence of damp and want of ventilation, along with penicillium mould. Diphtheritic sore throats were present in this house: may not the cause be looked for in the influence of these fungi?

Case 4.—A terrible calamity happened, in November 1885, to the family at the important farm of Hackthorpe Hall, an ancient large Elizabethan manor-house in Westmorland. I have visited the place since the occurrence, about which I am indebted for information to my friend Dr. Sanderson of Penrith; the events have been also specially referred to by Mr. Paget in his Annual Report on his Sanitary District. Three of the youngest children, aged about six, four, and two years respectively, were stricken with virulent diphtheria, and in one on whom tracheotomy was performed the trachea was found choked up with exudation; they all died within a week. A few weeks before these events it was noticed that the drinking-water standing in decanters in the bedrooms was cloudy and yellowish in colour, and deposited a slimy adhesive stain in the water-bottles. The house-supply was obtained from a tank, which had been formed about twenty years ago, in a field on higher ground about three hundred yards from the house. The tank was excavated in the boulder clay, and lined with loose stone walling; it was about sixty feet square, and from its average depth, might be computed to contain about a thousand cubic feet of water; it

was an open pond; it was fed by a main from a large field, five hundred yards distant, on the slope of the hill, which had been systematically tile-drained. The year before, this field had been converted from pasture into arable, and was being ploughed and prepared for turnip culture in the succeeding spring. For this purpose about thirty cartloads of farmyard manure had been carted out, shortly before the period we are speaking of, and deposited in a regularly formed heap as usual. It so happened that this midden was erected just over one of the main drains, leading into the large drain which conveyed the water to the tank; and when attention became directed to the water-supply, it was found that the liquid manure draining from the midden was passing into the main, and at the outlet into the tank, water was running into it of the colour of diluted port wine. Moreover—and this is the fact to which I would draw particular attention—at the date of the examination after the deaths, it was observed that not only was the water in the tank turbid and yellowish, but that there was a slimy, dark, purple-coloured mucor, growing on the stones on the edges of the tank, on both sides of the inlet of the main; that there was a large quantity of fine, hairy, colourless mycelial filaments radiating from tufts, diffused through the water, and adhering to particles of grass, straw, and leaves floating on the surface. These growths were observed to be present in the water in the tank for a considerable period afterwards; and a cattle-trough in the farmyard, which continued to be fed by a drain communicating with the main, for a long time after showed along its sides and edges a considerable development of this chocolate and purple-coloured mould, and hairy threads. I had not the opportunity of verifying with exactness the botanical species, but the character of the confervoid tufts was that of *Saprolegnia*, and that of the darker mould a *Mucor*. Now, in this outbreak, the water was indisputably the vehicle of the disease, and the consideration is, whether *materies morbi* was not carried in the cells of this fungoid vegetation, having been assimilated into its protoplasm during the growth in the noxious media.

Case 5.—During a visit to Alnwick in this recent spring of 1887, I accidentally learnt the particulars of a very curious incidence of diphtheria, which was related to me as bearing on the theory of the fungoid origin of the disease, which happened to be the subject of conversation at the time. My informant, himself a scientific observer and a naturalist, was the father of the young lady, who fell ill with diphtheria under the following circumstances. She was eighteen years of age, and at the end of October 1885 had a very bad

diphtheritic sore-throat, from which she was ill for three weeks. There was no diphtheria in the neighbourhood, and there was no assignable cause about the dwelling or surroundings. For three or four days before the attack she had been occupied with arranging for the decoration of a church for a harvest thanksgiving. She had been out for a whole day in Alnwick Parks, in the woods, searching under the trees, and picking fallen decayed autumn leaves, which she afterwards at home assorted and arranged, and out of the heaps fixed up the various usual devices. It is curious that at the time the father and mother attributed their daughter's seizure to this working and picking amongst the dead leaves in Alnwick Parks, which at that season would be teeming with fungus vegetation.

Case 6.—Dr. Williamson of Longwathby has kindly favoured me with the particulars of the condition of the premises in the hamlet of Salkeld Dykes, in which a family of three children were carried off by rapid diphtheria, two of them aged seven and five years respectively, in the month of August 1885, and one aged four years and a half, in June 1886. The water was from pipes which supplied the rest of the village, and was good. It was a small farm, three cows were kept, and a horse; and the manure from the stable and cow-house was in close proximity to the front door. The house itself, a small, two-storied stone-built structure, was on a low level; the walls were extremely damp on two sides, so that two small apartments were sometimes uninhabitable from damp and mouldiness in the interior. Within the house on one side of the passage was the place used as a dairy, 16 ft. long by 4 ft. 9 in. wide, with a small window half-way up the wall, fitted with perforated zinc, and very damp and dark. At the time of the fatal illness of the last child, two hams and two sides of bacon were hanging from the roof of this dairy, which were all covered with a very luxuriant growth of a bluish fungus with spongy cottony expansions two inches long. Underneath stood usually six bowls of milk. Both Dr. Williamson and Dr. Sanderson of Penrith, who attended this fatal case, were impressed with the suspicion that the presence of these moulds in the dairy may have had some kind of relation with the production of the disease.

Case 7.—I am indebted to Dr. Robertson of Penrith for calling my attention to the circumstances attending an outbreak of diphtheria severely affecting two in the family, the mother, and eldest child aged six years, at Huddlesceugh Hall, near Renwick, in February 1883. Dr. Robertson refers to this case in his annual report to the Rural Sanitary Authority in the year 1883, with the result of his investigation, as

follows: "The house itself is in every respect a most healthy residence, with large, airy rooms, and the most scrupulous attention is evidently paid to cleanliness; the family are in comfortable circumstances, and live in the most substantial manner as regards food; the vicinity of the house is in every respect most clean, and no nuisance in proximity to the house; the only true possible causes for the appearance of such a disease which present themselves to me are the water-supply, which I found to contain a trace of sewage, which must have been conveyed from a very considerable distance through porous subsoil; *or the influence of a fungus growth*, which grew with the most extraordinary pertinacity and exuberance in the rooms more especially occupied by those affected. The defective spouting in the house was the cause of some damp at the windows and window-seats of the rooms referred to, and from these situations this fungus, of a fine hairy appearance, developed to an extraordinary extent, notwithstanding its repeated destruction. This growth also appeared on the arched roof of the milk-house, on which moisture collected, and no doubt dropped into the milk, carrying with it this low form of growth." Dr. Robertson adds the remark, "the association of diphtheria and low vegetable growths similar to those referred to, has been observed, and by some believed to be a cause of the disease."

Case 8.—Again, Dr. Robertson in his Annual Report for 1885 refers to another case of well-marked diphtheria which occurred in Penruddock, in a cottage building used as a village shop and dwelling-house, with damp walls and flooring. Here also there was a persistent development of delicate thin hairy mould growing on the interior of the walls, skirting boards, and window frames. Dr Robertson mentions, "the only local condition which I could point to, as in any way likely to cause or aid in the production of the disease, was damp in the house itself"; and he adds, "this damp condition of a house, and particularly when it leads to the development of fungi on the walls, I have frequently observed in connection with the outbreak of diphtheria, and in this instance, as at Huddlesceugh Hall, all the other sanitary conditions were very good indeed."

I will now proceed to cite:—

Cases of Association of Diphtheria with Mould-Fungi from Personal Observation.

These observations embrace eight different outbreaks of diphtheria, occurring in five houses, amounting to twenty attacks, with two deaths. These cases of course exhibit a certain resemblance of conditions as to origin, and have been

expressly associated, with the design of assessing the import of the antecedent circumstances, which might have had concern in the causation of the disease. In this point of view, the series is, as it were, the knitting-up of thirty years of my experience of diphtheria, and is the pick from observations, of conditions of invasions, in hundreds of separate houses. Damp walls and moulds, of course, are not the only sources of diphtheria. Personal experience would readily furnish other groups of cases, which might elucidate other phases of propagation of *materies morbi*—cases originating from proximity to midden-steads, and privies—cases from impure wells, or from water-supply collected from drains, or springs which had become polluted from manure—cases from infection through attendance on the sick—cases from contact through fondling the patient, and by spluttering of sputa on those manipulating on the throat. That milk may be a vehicle for propagating diphtheria is certain, as is shown by several outbreaks in connection with dairies, as at Frimley last October; at Camberley, Devonport, and elsewhere. I have, however, never met with an instance of diphtheria from milk in my own practice; though my attention has always been alive to the influence of this medium, seeing that the very first discovery and proof of the transmission of infectious germs through milk, were furnished by the observations made by myself in Penrith, on two occasions, first in regard to Typhoid fever* in 1858, and secondly in regard to Scarlatina† in 1869, which have since been so abundantly confirmed.

CASE I. *Series of three cases of Diphtheria in one house.*—In this house there were four adult individuals and three female children. On the night of August 1st, 1880, one of these children, aged eight and a half years, sickened with headache and pyrexia. Next day she presented a carmine-coloured throat, over which was subsequently developed filamentous pellicles, and patches of white membrane adherent to the surface, with the glands about the maxilla swollen and tender. Parts implicated: both tonsils, uvula, and velum. The white patches were capable of being brushed off, leaving a red, tender surface underneath. By August 7th, the membrane had crumbled off. There were vibices on the skin, and an accession of pyrexia on eighth day. Slow recovery. A sister, aged nine and three-quarter years, began on August 3rd with premonitory symptoms, a brightly-reddened throat, with much pyrexia; subsequently, formation of white, adherent,

* "On the Communication of the Infection of Fever by Ingesta" (*Edin. Med. Journ.*, May 1858). By the Author.

† "On the Propagation of Scarlatina by means of the Milk-Supply" *Brit. Med. Journ.*, 1870). By the Author.

membranous pellicle over fauces and uvula, which finally broke away, leaving a red granular œdematous surface. Convalescent in five days. Finally, the youngest sister, aged seven and a half, began on August 8th; the filmy exudation covered both tonsils, and œdema of mucous membrane continued for a week after. All these cases were expressive of true diphtheria. None of the other inmates took the disease. Now it had not been a diphtheritic season; it was not a diphtheritic house; there had been no exposure to the disease; and no suspicion attached to the milk-supply, the water-service, or sewage. In casting about to discover the cause of the evil, I was struck with the condition of the bedroom in which these children had slept. A portion of the outside wall of this room was steeped in wet; paper detached; plaster reduced to a creamy consistence, the surface covered with the pellucid, glaucous filaments of a mould, more than half an inch long (*aspergillus*), in addition to which there was an ordinary mealy greenish mould. Moreover, spread over the surface of the wall, and closely adherent to, and growing from, the wet plaster and cement, there were numerous clusters of a pileate fungus, some in buttony excrescences, and others opened out into the toadstool form. This fungus was identified as the *Coprinus domesticus*. The history of the occurrence was this: up to a certain period it had been a dry house; a breakage had occurred in the roof and spouting above this apartment; on July 12th, nineteen days before the outbreak of diphtheria, there had been a great rainfall, which continued for a week; the wet poured into the walls; July 19th, roof was repaired; July 22nd, first appearance of fungus on the walls; August 1st, first case of diphtheria; second case, August 3rd; third case, August 8th.*

CASE II. *Diphtheria, death in three days.*—Thomas R., aged four years, a stout, well-nourished, active child; first noticed to be ill on December 26th, 1883, with hoarseness, cough, and hurried breathing. First seen on December 30th, and found to be in a state of suffocative dyspnœa. An examination showed that the tonsils and soft palate were enveloped in a dense layer of white diphtheritic exudation, which extended also to the buccal membrane. The respiration was hurried, difficult, and shrill; a clangous, barking cough, and hoarse, altered voice; there was evident extension of the exudation to the larynx and trachea. The case terminated on the following day, from increased obstruction and exhaustion of vital power.

The inmates were father, mother, and an older child; no

* For full details of these occurrences, see paper "On the Fungoid Origin of Diphtheria", *Brit. Med. Journ.*, July 2nd, 1881, by the Author.

further extension of the disease. This was an isolated case, there was no diphtheria prevalent in the neighbourhood. The house was a solitary labourer's cottage, of four rooms, two stories high, situated on a height; on a dry sandy soil, on a substratum of the Permian sandstone. It was a comparatively new building, constructed of the sandstone of the quarry, closely adjoining. A short time previous to the illness of this child, there had been violent storms of wind and rain, in consequence of which there had been a great leakage of water through the walls of the house which faced the storm. The bedroom upstairs, in which the child had slept, was a small room, 10 ft. square, and 7 ft. high, with a window opening about 3 ft. square, and a small fire-place, which, however, was blocked. The walls were of plaster; they had never been papered, but were coloured with a blue lime-wash. The walls were always more or less damp in the winter season, but they were never so bad as they had been since the recent storms, after which they were running with water. At the time of my inspection, they felt quite damp to the touch, they gave out a musty smell, and were covered with a mealy, greyish-green mould; and articles of clothing, after hanging in this room for four or five days, became covered with mildew. The adjoining bedroom, which is rather larger, with a small window, but no fire-place, presented a still worse aspect from the effects of damp; the walls were simply covered with a growth of mould, and an old dress was shown to me, which had been hanging in this room for a week, of which the penicillium fungus had taken complete possession, and on which it was ripening its fruit-heads; an old piece of carpet which had lain in a closet, had become thoroughly rotten, from the penetration of mould. But another unsanitary element of an unusual character was discovered in another small room, which abutted on the sleeping chamber. Here were found, hanging about the room, and on the walls, a quantity of cat-skins, over a hundred in number; most of them were covered with mould, adhering both to the fur and to the skins; the whole place was charged with dust, and a putrescent effluvium. The father was a gamekeeper on the estate, and he killed the vermin, and wild or prowling cats, and skinned those animals, and preserved the peltry for sale; and it had accumulated in this room for some months.

Here the only apparent exciting causes of diphtheria were, the leakage into the walls, the wet plaster, and, it might be, the great development of mould-vegetation on the premises, on the different feeding-grounds mentioned.

CASE. III.—Charles V, aged eighteen, fair hair, light com-

plexion, fine delicate skin; was visited on January 30th, 1879, and found to be suffering from a diphtheritic sore throat. The exudation was ash-coloured, pulpy in consistence, and involved the right tonsil, with patches on the left; subsequent extension over uvula, left tonsil, and margin of the velum. The membrane presented usual diphtheritic characters, and was frequently peeled off, and detached by brushing. Considerable pyrexia, and much local trouble and discomfort, pain and engorgement of the maxillary glands. The local manifestations lasted for about six days, and were succeeded by deafness in left ear, and lowness and debility continued for eight weeks.

The inmates of this house, which was situated in one of the streets of Penrith, consisted of father, mother, and seven children, two older and four younger than the patient, and one female servant. None of these took the disease. There had been no prevalence of diphtheria in the district for a long period; this was truly an isolated case; no unsanitary conditions could be discovered about the house or surroundings. However, an inquiry into the history and occupation of the patient, in the week preceding his illness, disclosed the following fact:—This youth had a fancy for pigeons, a number of which birds he kept in an attic, over some unused buildings, quite away from the dwelling-house. Four days previous to his illness, he had been engaged all day working in this loft, cleaning out the pigeon-holes, brushing, sweeping, and putting up the nesting-shelves. The place was damp and dark, full of dust and dirt, and smelling of mouldiness. On returning home, he spoke of the dirty job he had undertaken, and complained of having been half-choked with the dust; he himself attributed his attack to his overheating, and exposure on this occasion. There was mould on the walls, and rotten woodwork, and the pigeon *débris* afforded a habitat to some of the familiar mucors. Order of incidence: exposure, January 24th; commencement of diphtheria, January 29th.

The consideration of the following group of cases involves no less than five outbreaks of well-expressed diphtheria in one family, at distant periods, and, in point of fact, gives the life history of these children in relation to this disease. Moreover, these attacks arose during the occupancy, not of one residence, but of two houses standing under quite opposed conditions, in regard to aspect, subsoil, and elevation. I will divide the narrative, therefore, into two series.

CASE IV. *Cases occurring in first residence.*—This takes us back to the year 1870. At that time, the family consisted of

father and mother—farmers, both young and healthy—a household servant, and five children. As we shall have to distinguish the different members, I give the children's names and ages at this date, as follows:—Elizabeth, aged eight; John, seven; Isaac, five; Richard, three and three-quarters; Jane, two years.

On October 7th, 1870, summoned at night to see Richard, who presented symptoms of a commencing croupy affection, and next day an investment of white exudation was detected on the fauces, which subsequently increased in extent, and grew more opaque and leathery. The child suffered extremely; there was croupy breathing from the first, and it continued to the end; the child sank on the fourth day. Within two or three days Elizabeth and John sickened, and had diphtheria in its typical form, confined, however, only to the palate and fauces; and they made a good recovery. On October 27th, the youngest child, Jane, took the disease, showing the usual distinctive membranous patches on the soft palate and adjacent parts; she was confined to bed for a week, and recovered, after a good deal of distress, from the mechanical obstruction to breathing and swallowing. Continuous attendance on these four cases, from October 17th to November 12th.

Here, then, in this household, four children were struck with diphtheria within the space of two weeks, but it spread no further. There was no prevalence of diphtheria at this time; in point of fact, after the subsidence of the first great epidemic wave, which occurred in 1859, 1860, and 1861, diphtheria had become, for some years, comparatively rare in that part of the country as a spreading disease. It could not be perceived at the time that any particularly unsanitary conditions existed in the surroundings of this house, beyond damp, and a soakage of the soil from recent rains. It was a modern stone-built farmhouse with six rooms, clean, isolated, standing on high ground on Clifton Moor near Penrith; the soil was the boulder clay, which overlaid the limestone rock. The cause of this outbreak remained then, and for years after, a mystery.

CASE V. *Subsequent outbreaks in this family.*—We proceed to the year 1879: the family had removed from Clifton in 1873, and had entered on the occupation of another farmhouse, about seven miles off, in the parish of Dacre, in Cumberland. Meanwhile, other children had been born; Eleanor, in 1871, William, in 1873, and Thomas in 1875; so that at this date, they were seven in number.

Series A.—In January 1879, Thomas, aged four years, was

seized with a throat affection, with the exhibition of white pellicles over the tonsils, and faucial-arch, with subsequent croupy cough and breathing; it was regarded as diphtheritic, and treated accordingly with topical brushings. The condition of the child was such as to cause much alarm, but it recovered after a struggle of a fortnight's illness.

Series B.—On March 17th, 1880, the eldest daughter Elizabeth, aged eighteen years, sickened with lapitude and pyrexia, with subsequent development of diphtheritic exudations on tonsils and fauces, with pain and swelling of the maxillary glands; the membrane was a white milky film, with branching filaments, forming an adherent coating, but did not thicken to much extent. The illness was succeeded by rather persistent debility and anemia. The little brother Thomas, who had gone through the bad attack of diphtheria fourteen months before, was seized on the following day, with precisely the same symptoms—with the white exudation on the throat, but with less severe croupy manifestations. Within the week another case occurred: Isaac, aged fifteen, became ill with throat symptoms, white patches on both tonsils, difficulty of swallowing, enlargement in the sub-auricular region. The diphtheritic coating was renewed several times, after swabbing with perchloride of iron. Finally, a fourth case began, within the next week: John, aged seventeen, was seized in a similar manner, and presented the usual throat exudation. They all recovered.

Series C.—On July 2nd, 1881, William, aged eight years, who had escaped the preceding invasion, sickened rather suddenly, and was visited at night. It was seen that diphtheria was being displayed in broad, white, milky films, running over the tonsils and uvula, with high redness and swelling. Notwithstanding brushings, these patches continued to be reproduced for two or three days, after which they wore off, leaving puffiness of the subjacent tissues, and some swelling on the sides of the neck. Within a day or two, Thomas, now six years old, who had gone through diphtheria twice before, and who came to be called the *croupy* child, became again affected in the same manner as his brother, with an expressly typical exudation, and spreading over the tonsils and fauces. Both these children were ill about a week.

Series D.—On August 11th, 1881, Jane, aged thirteen, one of those who had diphtheria at Clifton, when two years old, began with white patches on the throat on both sides. She had no initiatory symptoms, and was out in the turnip-field the day before. The throat was swabbed, she was visited for four days, after which she was well. Her sister Eleanor, aged

ten, who had hitherto escaped the complaint, became affected on the 14th with stiffness and swelling in the angles of the jaw, and exhibited small white patches on the fauces; it was slight, however, and she was better in a day or two. Finally, the mother, Mrs. B., who had all along preserved a freedom from this throat affection, and who had been quite well the day before, complained on the 31st of general lassitude and indisposition, and soreness and dryness in the throat, which on examination showed a white patch on the left tonsil; next day there was a recurrence of a slight exudation, but it disappeared after the second swabbing.

Incidences of Diphtheria in this Family.

1870.	1879.	1880.	1881.	1881.	Total.
October.	January.	March.	July.	August.	
4 cases out of 5 children.	1 case out of 7 children.	4 cases out of 7 children.	2 cases out of 7 children.	3 cases out of 8 inmates.	14 cases. 1 death.

One individual had the disease thrice; three had it twice; five had it once.

The incidence of these outbreaks of diphtheria in this house in little more than two and a half years attracted attention, and accordingly, on July 9th, 1881, I made a special inquiry as to the condition of the premises. It was a substantially-built farmhouse, with surrounding offices, standing by itself on the slope of a limestone hill, with good natural drainage; four good sleeping apartments on the upper floor abutted on a landing. No fault could be found with the structure, nor with water-supply, or privy arrangements. There was no constant antecedent circumstance about the place, which could account for these events. But in going through the house, I was taken into a room on the sleeping floor, and I was amazed to find it nearly filled with wool. This wool was not sheeted, but the fleeces were loose, each being bundled up as usual and piled up, so as to occupy most part of the room, from the floor nearly to the ceiling; there must have been several hundreds of fleeces. The explanation of this accumulation of wool was the following: the farmer had entered on the farm eight years ago; for the first two years, he sold out annually his crops of wool; after which time, in consequence of a falling market for the product, he was induced to keep his clips from year to year, storing them one above another in this room, which now contained the crops of six years—

that is, since 1875. The air of this room was full of dust, and had a fusty, mouldy smell, and though the walls were dry and free from vegetable growth, I have no doubt that some forms of mould existed amongst the layers of wool. It at once struck me that the storage of wool within this house had some substantive connection with the elements of mischief. Let us see to what extent this suspicion was borne out by the internal evidence of the sequence of events. There were two bedsteads in this room. It was first occupied as a bedroom by a man-servant, who was hired in November 1878; he was the sole occupant until August 1879, when he left the service; he never had any illness whilst he was there. Some time after this man left the house, the two sons, John and Isaac, aged fifteen and seventeen years, were put into this bedroom. These lads were two out of the four who took diphtheria in March 1880, the third individual being Elizabeth, the oldest daughter, whose office it was to clean and sweep this room; the other case, on that occasion, having been the five-year-old child Thomas, about whose exposure there was nothing definite. Further, it appeared that six weeks previous to the outbreak of diphtheria, in July 1881, a change was made amongst the boys as occupants of this wool-room, and William, aged eight, and Thomas, aged six, were put into these beds. It is significant to observe that these two boys were the sufferers on this occasion, whilst the rest of the family escaped. But the tale does not end here: how about the bad Clifton outbreak in 1870? Now, at last, there was an unexpected gathering up of evidence, presumptive of the source of evil. It came out, that at the Clifton farm it had been the usual practice at first to sell the wool every year, but that in 1870 two years clips had been allowed to accumulate. This wool was also stored in the house, in a small room upstairs, opening on the bed room landing; there was no bed in this room, but the children had access to it. Thus, from these facts, two issues are evolved, viz.:—(1) when the wool was sold off each year, diphtheria was absent; (2) when the wool was stored within the premises above two years, diphtheria did occur. Setting aside for the moment the theory of the influence of moulds, the presumption is strong that the storage of the wool had some concern with the advent of diphtheria in these cases. The whole history of the occurrences, the sequence of the circumstances, the incidence of the disease on individuals in proportion to the amount and recentness of their exposure, involve events which would be marvellous if treated as coincidences, and which would be totally at variance with the probability of chances.

Remarks.

In comparing the conditions of invasion in all the cases, I take the common denominator to have been the exposure of the patient to the spores of certain common fungi, growing on various media. In case No. 1, the feeding material consisted of decaying animal and vegetable matter, sodden with wet, glue, size, paste, paper, and the hair and plaster on the walls. In No. 2, the moulds found a habitat on the wet walls, the animal skins, and old woollen clothes; and in case No. 3, amongst the *débris* of the pigeon loft, and the feathers and exuviæ from the nests. In cases Nos. 4 and 5, a fertile nidus was afforded for the growth of mould in the fleeces of wool, kept for years heaped up in one room.

As to the botanical identification of the mould-fungi, which were observed in all these cases, I regret I cannot be positive about the determination of the species. The pileate fungus mentioned in case No. 1, which took such inveterate possession of the wall that it grew there for months after, was kindly determined for me by the Rev. M. J. Berkeley, as being *Coprinus domesticus*; but I do not attach too much significance to the presence of this particular cryptogam. The mould-fungus associated with the *Coprinus*, as suggested by the above eminent Fungologist, was either a form of aspergillus, or may have been what Sowerby calls a "Fibrillaria". I could not specify the fungus growing on the cat-skins, but the growth on the walls and clothing was in whitish-grey tufts, studded with dew-like drops, the commonest of moulds, the *Penicillium glaucum*. The fungi in the pigeon loft were somes modes of mucors and aspergilli; in allotting a cause in this instance, allowance must be made for the fact that pigeons are subject to diphtheria,* within the beak and posterior part of the palate, which occasionally prevails as an epidemic, and sparrows are known to be subject to the same affection. There was no suspicion here, however, of any such incident.

If we conceive that the strings of micrococci, and other fungoid organisms found in the exudations of diphtheria, be not of pathogenic import, but merely adventitious and secondary growths in decomposing structures, it would not be worth while to pursue the inquiry any further. But the strength of opinion is quite in the contrary direction.

The researches of Oertel, Nasiloff, Letzerich, Ebert, Talamon, and others, all tend to show that diphtheria is a

* "Diphtheria in Dove-house Pigeons," Edward Crisp (*Path. Soc. Trans.*, vol. xxv, 1874). Dr. Bristowe records similar diseases in uncaged doves (*Ibid.*, vol. xiii).

true mycosis, and that the mycelium, composed both of spheroids and cylindrical rods, inclosed in the layers of the pseudo-membrane, penetrates into the sub-mucous tissues, infiltrates into the neighbouring glands, circulates in the blood, and forms metastatic foci in various viscera. This warrants us in espousing the belief, that the disease depends on the introductions from without of some fungoid organism endued with virulent power.

Therefore we may now proceed to work out the probabilities of common moulds being connected in some way as agents in the process; and it may be permissible to derive evidence from analogy of what obtains in other affections showing points of resemblance to diphtheria, if such evidence in essentials be applicable to the quest for truth in this problem. Thus it is reasonable to compare diphtheria with some of the parasitic affections which infect men and animals, and with some which infest, as epidemic scourges, both the vegetable and animal kingdoms, in so far as they may consist in their essence of vegetable growths, pervading with their fungal threads, more or less deeply, the tissues or structure of the individual.

But there is strong reason to believe that many of the different structural forms and species of these fungal productions may be but conversions of elementary states of the yeast and mould-fungi—the *oïdium* and *penicillium*. For fungologists have found, that in the life history of these micro-organisms, there are manifested alternations of development, a discontinuity of form, and a variableness of generic character, induced probably by special conditions of food and habitat; and that it is possible that many of these different homologues may really be of identical origin.

The white patches of thrush, aphthæ, muguet, pseudo-diphtherite, depend on the development of the *oïdium albicans* of Robin, which according to Hallier, is nothing else than the *oïdium lactis*, the fungus of the acid fermentation of milk. The identity of *achorion*, and other parasites affecting the skin of man and animals, with *aspergillus glaucus*, was long ago shown by Dr. Lowe*; Gräwitz† describes the parent form as *oïdium lactis*; and, more recently, Malcolm Morris and Henderson,‡ have demonstrated by cultivation, that the spores of ring-worm (*Trichophyton tonsurans*), produce the branching septa formation and fructification, which are identical with those of *penicillium*, and that the spores of the second generation produce ring-worm on the human skin.

* *Trans. Bot. Soc. Edin.*, 1858. † Virchow, *Archiv*, vol. 70.
‡ Morris and Henderson, *Journ. Roy. Micros. Soc.*, 1883.

Investigations render it probable, though the whole of the facts may not yet be fully established, that the disease of the house-fly, the *empusa*, alters its whole character when immersed in water, and forms the confervoid tufts of *saprolegnia*, which infest the salmon and gold-fish; nor is this all, for there is reason to suppose that this fish parasite, when implanted on the silk-worm, forms the *muscardine*, or *botrytis*, which again may be resolvable into the *oïdium* of the vine and potato blights.

Most recently Zopf has warmly espoused the doctrine of pleomorphism amid the Fission-fungi, and views the various shapes of bacteria as but steps of development of the same micro-organism, and that there are stages of intermediate forms between leptothrix, bacterium, and coccus, and even transformation of the threads into spiral vibrio, and spirochæte forms, resulting from the nature and composition of the nutrient fluid.*

That common moulds, even in a non-pathogenic condition, are capable of artificial growth within the tissues of living animals, is a fact borne out by the assertion of Leber,† that the spores of *aspergillus glaucus*, introduced into the eye of rabbits, in the juice of fruits, germinate freely and equally there, at a temperature of 35° to 37° C. Indeed, there is a ready-made experiment in the disease of the Madura foot in India, which is a true mycetoma, affecting the tissues of the extremity, even to the bones. This tumour arises from the spores of a cryptogam, (which has been referred, by the Rev. M. J. Berkeley, to a state generically allied with the mucors or moulds), which grows in the hot season in India, effecting an entrance into the skin by a small wound, such as the prick of a thorn.

But we know full well that these yeast and mould-spores which pervade our houses, and are present everywhere around us, are harmless to us and living animals. How is it, then, that they may acquire disease-attaching power? Can a harmless aspergillus change its physiological action, and become potential for mischief? Can an innocent bacterium become a deadly one; and, if so, by what process? It is in a belief that such changes do occur that I seek for the determination of the argument. Dr. Gräwitz maintains that he has obtained, by cultivation of the moulds aspergillus and penicillium, products of two distinct orders, morphologically identical, but one of them in its constitution pathogenic and very poisonous, whilst the other may circulate with

* Zopf, *Die Spaltpilze*, 1885.
† *Berl. Klin. Wochenschr.*, 1882.

impunity through the blood of animals. Similar results have been obtained by experiments on rabbits, by Fessinger, who goes so far as to suggest the possibility of enteric fever being generated by ordinary moulds, the spores of which may have become endowed with pathogenic properties on account of spontaneous cultivation in noxious media.

Professor V. Nägeli, of Munich, is a very strong advocate of this sporting tendency amid micro-organisms. He maintains that when a harmless form of bacterium is brought under suitable conditions, it may become the origin of infectious diseases; and, further, that it may through generations maintain this power, and when again placed under different conditions, it may change into an inactive form.* Büchner, the pupil of Nägeli, claims to have confirmed these experiments, and to have succeeded in changing the bacillus of charbon, of previously deadly power, into a perfectly inactive and impotent bacillus, which in morphological respects appeared identical with the B. subtilis (Cohn) of hay infusion. He also thinks he has succeeded in transforming, through cultivation in albuminous fluids, the hay bacillus into one possessing virulent properties. Conflictions of opinion, of course, do exist amongst bacteriologists; and these results of Büchner have been criticised and opposed by Koch, Klein, and others, who support the views of Cohn, on the permanence of generic forms.

Again, to go a step farther, another fact bearing on the subject has been demonstrated from Lichteim's experimental inquiry on "Pathogenous Mould-fungi,"† viz., that some which are innocuous to man, are pathogenic to rabbits and other animals; thus, amongst the aspergilli, he has found two pathogenic forms, one yellowish, *A. flavus*, and one with greenish tufts, *A. fumigatus*, and possibly another with brownish-black conidia, *A. niger*, which exists saprophytically in the human intestine, in the passages of the ear, and in other localities, but which, when introduced into the vascular system of the rabbit, produce metastatic foci in the various organs, and viscera, and deadly results.

Thus, though we have approached the question of the etiology of diphtheria from the clinical aspect, or from the outside, as it were, yet it would seem that these speculations from bed-side experiences are in some measure sustained by the experiments of the laboratory.

If it be true then, that the micro-organisms involved in the ordinary process of putrefaction, and the common moulds

* *Die Mederen Piltze*, etc. Munich, 1877.

† Report in *Journ. Micros. Soc.*, vol. iii, 1883, p. 250.

attacking decaying organic matter, usually innocuous and harmless, shall at other times become active agents in originating an active mycosis in living tissues, under what conditions do they acquire their pathogenic properties? The explanation of this may involve considerable modification of the views at present dominant, regarding the specificity of individual forms, as primary elements in originating septicæmia and zymotic affections. It may be an affair of the soil in which they grow—a special infectivity in the protoplasm of the organism, be it a bacterium, a bacillus, or an aspergillus spore,—a chemical organic, but unorganised poison, elaborated from putrefactive media, or derived from a living host, matured as a species of ferment in the walls of the microphyte during stages of its growth, and, along with the germ, capable of being multiplied, and of determining characteristic products in living tissues; so that we may come to regard the germs of various forms of fungal life, now reputed as having exact relationship with special zymotic diseases, to be but carriers of infective material, under fortuitous conditions, whilst at other times these same micro-organisms may be innocuous.

The argument which we have followed in attempting to demonstrate the Proposition may be epitomised thus:—

1. The *Premises.*—Citation of cases illustrating the concomitance of diphtheria with the presence of common moulds, growing under pecular conditions, in the presumable absence of other attributable causes.

2. That the essence of diphtheria consists of fungoid vegetation.

3. That other diseases analogous to diphtheria, in essentials of fungoid character, may arise from morphological states of common mould and yeast fungi.

4. That common moulds in a non-pathogenic condition can be made to grow in living animal tissues.

5. That innocent forms of mould may acquire, in noxious culture media, toxic properties.

6. The *Conclusion.*—That many of the cases herein referred to may have had their origin in the implantation on the throat of spores, or byssoid mycelium of common fungi, which may have acquired such virulent properties that they were enabled to overcome the resisting power, or *vis naturæ*, of living tissues.

In presenting this deduction, though possibly it may be deemed as yet a speculation waiting for further proof, I hold it to be nevertheless warrantable, seeing that it does not run counter to, nor stand quite outside, the present lines of the teaching of biological research.

SOME EVIDENCE RESPECTING TUBERCULAR INFECTIVE AREAS.

By ARTHUR RANSOME, M.D., M.A., F.R.S.

(*Read: May 11th*, 1887.)

THERE are strong reasons for believing that whilst tubercle travels infectively through the body, and is derived from infective particles contained in air rendered impure by respiration, yet in this climate that it is very rarely produced by direct infection from person to person. It seems most probable, in fact, that for the active propagation of the disease, some increase in the virulence of the organism must take place outside the body, this intensification of its power being most commonly produced by the presence of animal organic matter in the air, in other words, by the absence of efficient ventilation. The favouring influence of a damp subsoil is also very distinct, and is probably due to the quality of the ground-air arising from such soil. With such premises as these it might fairly be anticipated that certain distinct areas of infection would be found in both town and country, though most abundantly in crowded, ill-ventilated houses in the low-lying, badly-drained districts of towns.

Perhaps the best examples of infecting areas are to be found in the records of public institutions, such as workhouses, prisons, orphanages, etc. Hirsch, in his *Handbook of Geographical Pathology* (*Syd. Soc.*, vol. ii, p. 632), gives a long list of such instances relating to scrofula, and others relating to consumption in vol. iii, p. 222.

Dr. Parkes, also, in his work on Hygiene (6th ed., p. 134), gives some additional examples; and Laennec, in his classical treatise on Phthisis, records one notable instance of the kind, in which the population of a religious community of women had been changed two or three times from phthisis, with the notable exception of those who had charge of the garden, kitchen, and infirmary. The records of death from phthisis in the army, from unhealthy barracks, in all quarters of the globe, and similar returns from unhealthy ships in the navy, and in the mercantile marine, show the same thing. I believe all these accounts to be instances of bacillary infec-

tion from the fostering influence of bad air or bad drainage, or both.

But if the theory is sound, it ought to be possible also to find examples of breeding-places for the disease, "phthisis nests" as they might be called, in the narrow streets and courts, and even in individual houses in our towns.

The inquiry is one of some difficulty. It is thus not always possible to separate the effects of hereditary tendency to the disease from those of unhealthy areas, and the question of the possibility of direct contagion also enters the arena of discussion. With regard to the first-mentioned source of difficulty, however, it must be remarked that, according to our present knowledge of the matter, a hereditary tendency to consumption, or the scrofulous diathesis, as it used to be called, means little more than a peculiar vulnerability of the system by the specific organism, and hence it simply intensifies the operation of favouring external circumstances. It will thus assist both direct and indirect infection.

The possibility and probability of the direct conveyance of the disease, from patient to patient, is of more importance in regard to our immediate subject, seeing that many of the cases supposed to be due to local conditions and indirect infection might just as reasonably be ascribed to direct infection. I would also grant at once that there is nothing antecedently improbable in the theory of direct infection, although such infection would be likely to be difficult in persons in a healthy condition.

Professor Koch has shown that the bacillus of tubercle needs for its development: 1, a suitable medium containing organic matter, such as ox-blood serum, or the Japanese gelatinous substance, Agar-Agar; 2, a temperature of between 87° and 106°; 3, a sojourn of a week or more under these conditions.

It is probable that the last-named condition accounts for the immunity of the healthy lung to its attacks. The bacillus can obtain ready access to the air-passages of us all, seeing that it is probably often contained in the dusty atmosphere of towns or in the impure air of meetings at which some consumptive or other may be present. It would also find both suitable pabulum and a suitable temperature within the human thorax, but from healthy lungs it would soon be ejected, either entangled in mucus, or passed out by the action of the cilia lining the air-tubes. Such a fate need not necessarily befall it, however, if the lung had lost its natural elasticity by cramped and confined positions of the body, or by attacks of inflammation of the substance of the lung; and we know that such attacks are common precursors of true phthisis. There

would be nothing wonderful, therefore, in the direct engrafting of tubercle upon such lungs, and yet I believe that such direct transference of the disease from one person to another is a very rare event in this climate.

I have elsewhere given my reasons for coming to this conclusion, but I may perhaps be allowed to give their substance here.

1. We may note, as the result of a special inquiry conducted by the Collective Investigation Committee, extending over a period of many years, that, out of some millions of cases of phthisis that must have occurred during this time, there are only a few hundreds of supposed cases of infection deemed worthy of record, and many medical men expressly state that they have never seen a case of direct infection.

2. Upon analysing these cases by means of Dr. Longstaff's formula, given in the *Collective Investigation Record*, we find that the number recorded of cases of phthisis in husband and wife, within ten years, falls much short of the number that would probably have occurred in the practice of the observers supposing there to have been no infection at all in operation.

3. In most of the cases given in the *Record*, the persons supposed to have received direct infection lived under similar conditions, and for the most part in the same houses, and these conditions are often noted as having been very unhealthy. The infection is just as likely to have been indirect instead of direct, *i.e.*, from person to person.

4. If phthisis is directly contagious, it is remarkable that it should chiefly be contagious on certain soils; and, further, it is difficult to see why drainage of the land should affect its contagiousness.

I have nowhere found any satisfactory proof of direct infection in a well-ventilated house, or even in the well-ventilated wards of a consumptive hospital, and this in spite of close contact, as in the attendance of a wife upon her husband, or in the nursing and sleeping together of near relatives and friends; such an immunity, then, cannot be due merely to the disinfecting action of fresh air.

I may mention incidentally a striking example of the influence of soil with which I became acquainted during the past year. The committee of the Hospital for Consumption, with which I am connected, had occasion to seek for a site for new wards. I had been struck with the freedom from the disease enjoyed by a portion of the locality in which I myself live, and it occurred to me that I might ascertain how many cases originated in the different parts of the district. A great portion of it is composed of deep, porous soil, but

it is surrounded by boulder-clay, the result of glacial drift; and a great part of Bowdon, and parts of Dunham and Altrincham, are built upon a thick bed of sand, in many places over 100 feet in thickness. The climate is thus rendered more temperate, and the air and soil drier.

After the wettest weather the paths speedily become dry, and the basement story of a house is often as dry as its attic. It has the further advantage that it is virgin soil. The sand is as pure and free from organic matter as in the days when it was deposited by ice-floes, or was silted up by the estuary of the Mersey. No house is ever built upon freshly-made ground, or on pits that have been filled up with refuse. The locality is well sewered, and has a plentiful supply of good water. Moreover, the inhabitants are for the most part well-to-do people. Out of 2,559 of population at the last census, only about 500 are poor, and live on the low-lying clay lands that surround the sandy downs upon which Bowdon is built. The remainder dwell in well-built, salubrious houses; they are well fed, and comfortable in their circumstances.

It will thus be seen that such a community are in a position peculiarly well-fitted to preserve them from attacks of tubercular disease. I was, however, hardly prepared for the result of my inquiries.

I obtained from the superintendant registrar of deaths an extract from the death-register of all the deaths from diseases of the lungs occurring in Bowdon, in the ten years 1875-84. Of these, twenty-two were from phthisis, but eleven of them took place in the low-lying clay lands before mentioned, and nine of the remainder were found to have contracted the disease before coming to Bowdon. This leaves two to be accounted for, and one of these was a gentleman connected with the City Mission in Manchester, who was, therefore, constantly obliged to attend crowded evening meetings in different parts of the town. The other was a merchant's clerk, who went to town at eight every morning, and did not return until 7 p.m. No woman or child died of the complaint; in other words, the disease did not originate in any of the stationary population, resident during the ten years upon the sandy portion of the district.

It is certainly difficult to see why the condition of the soil should so greatly affect the distribution of the disease if it is mainly propagated by direct infection. Another argument, which I owe to my friend Dr. W. H. Broadbent, may be found in the frequency of cases of one-sided phthisis.

It is on these grounds that I venture to affirm the doubtfulness, or at least the extreme rarity, of direct contagion in

phthisis. The very cases that are claimed by the contagionists as evidence of direct infection may therefore often be taken as proof of the existence of infected areas; and thus I think that I am justified, in support of my theory, in calling attention to any instance in which phthisis has prevailed especially in certain localities, in certain streets, and even in certain houses.

My own attention was first called to the subject by some cases occurring in my own neighbourhood. One striking instance was that of a whole family of six members carried off by the disease in one house, the mother, an old woman, alone surviving. In another family, again, four or five children were thus disposed of, one of them, a son, dying in another house after he had contracted the disease at home; but a daughter who was in service escaped altogether. In two other instances several members of each family died of the disease within short periods of one another.

The important feature of all these cases was that they occurred in small, badly ventilated cottages situated upon clay soils. The *Collective Investigation Record* contains a number of similar observations.

Dr. Bampton, of Plymouth (No. 193), supplies three such instances, in one of which a family, consisting of a mother and nine children, all died of phthisis, the father remaining healthy; several of the grandchildren also died of tubercular diseases, and not in the same house.

Nos. 159, 165, 168, 178, and 188, are also very striking cases. In most of these instances it is expressly mentioned that the houses in which the patients lived were small and ill-ventilated.

One observer, Dr. J. S. Dewar, of Arbroath, remarks that in all his cases (No. 166) "the patients lived in small, confined houses, and slept in the 'box-beds' in use in Scotland." "During twenty-five years," he says, "I have not seen one case of contagion in the airy houses of the well-to-do."

I think one may reasonably doubt, therefore, whether the contagion was not indirect, and due mainly to the intensifying power of the atmospheric impurities. In two or three instances the virus certainly seems to have been introduced from without into houses previously entirely free from the disease.

Thus: No. 188. I. McI., aged 21, student, son of healthy parents, both living, no family history of phthisis; took ill with a cough while studying in Glasgow in 1870, neglected it, came home at the end of the session, cough got worse, found both lungs tuberculous, died of phthisis in nine months.

Had one sister, 19, and one brother, 17. The former a perfectly healthy girl, nursed her brother closely, got ill shortly after his death, and was dead in five months after him of phthisis. The brother, who slept with I. McI. during part of his illness, and wore his clothes after his death, showed symptoms of failing health before the sister died, and in about eighteen months died of phthisis. Parents still living in 1883.

No. 194. In 1862 a servant came home to her mother (a widow with three sons and two daughters, all grown up, father dead of epithelioma) suffering from phthisis. The house, consisting of two rooms and an attic, and lying under the brow of a hill on its northern aspect, was ill-ventilated and worse lighted. By the end of 1868 the only survivor of this family was a thin, delicate girl, who took little or no part in the nursing. They all died of phthisis, the mother dying last, between fifty and sixty years old.

No. 196. A young man of the Indian navy came home suffering from phthisis. In a few months two of his sisters were taken with the same complaint, and died. A third sister married, and soon afterwards died of the same complaint; the young man also died. Later on the father was similarly affected, and died. After his death the widow became phthisical, and died also. Four years covered the whole outbreak, that is, from the arrival of this young man from India. The father was originally a very strong, healthy man, and all the children healthy up to about twenty or twenty-one, or even later. One sister still lives, and is now between forty and fifty.

No. 255 is a somewhat similar case.

The London Medical Record for July 1884, quotes a case by Dr. Kempf, of Louisville, tending towards the same conclusion, namely, that the disease may be introduced into a dwelling from without, and also that it may be eradicated from it. I do not think it proves anything more, for I should greatly doubt the statement that the convent in question was well-ventilated. The story is, however, as follows:—

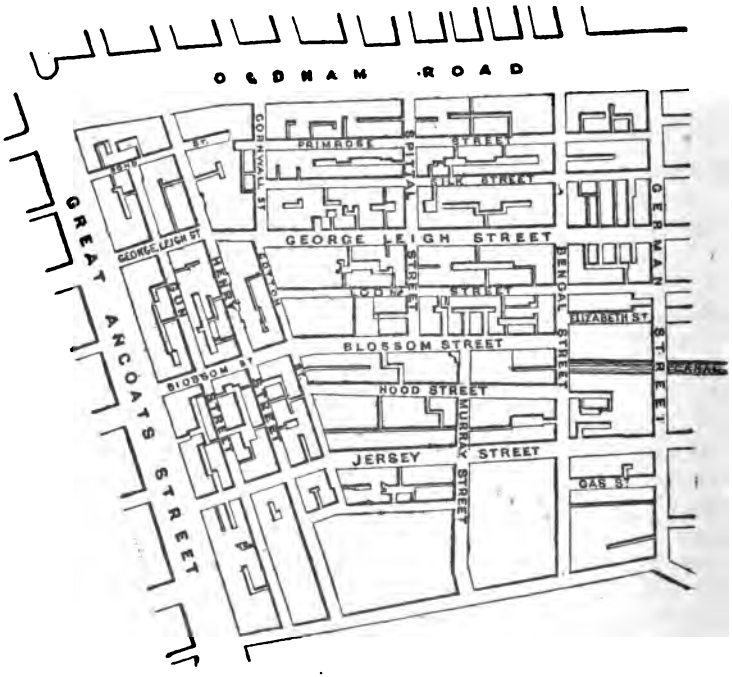
In 1880 the Sisters' Convent, in the village of Ferdinand, was entirely free from consumption; it is well ventilated, is high and dry, and is well drained. In the autumn of 1880, a girl of 18 was found to be consumptive. She continued to sleep in the general dormitory. One sister after another now commenced with similar symptoms, and in four months after the first case began there were nine cases in sisters who had been thought to be exceptionally healthy. Four sisters died in the course of a year. After complete isolation of the sick, the epidemic was stopped.

These cases in themselves would be sufficient to show the probability that phthisis may become, at any rate for a time, epidemic in certain houses.

It seemed to be probable also that a research into the intimate history of some of the unhealthy districts of our large towns would supply similar evidence, and I have therefore undertaken an inquiry into the distribution of phthisis in certain parts of Manchester and Salford. In this inquiry I have been much assisted by the admirable manner in which the records of mortality are kept in both these towns, and the mode in which they are broken up, for statistical and other purposes, into small and manageable areas.

I have also to thank the officials of the health offices of the respective corporations of these towns, and especially Mr. Dawson, of Manchester, and Dr. Tatham, Medical Officer of Salford, for the readiness with which they came to my assistance, and for the excellent mortality tables that they had prepared for me of the districts in question.

In Manchester, almost at haphazard, I selected a small part of Ancoats, of which a map is here given.



The following description of this district is given by Mr. Leigh, Medical Officer of Health, in his *Report*, published in 1884:—

"Nearly the whole of the houses in the district were built before the year 1830. There are a great number of back-to-back houses in all parts of the district. The width of the streets ranges from 30 feet to 4 feet, the greater number of them being 16 feet or 17 feet wide. The passages vary in width, and are only 2 feet 6 inches wide in some instances; in many cases the backs of the houses are too near to each other.

"The streets are for the most part paved and sewered. The houses, as a rule, are without cellars, and have no ventilating spaces under the floors. They rise directly from the ground; the outside walls are 9 inches thick, and the inside walls are 4½ inches or 3 inches thick. They smell fusty; the ceilings of some are only 6 feet from the floor; the timber of many is in a state of decay. Houses in such a condition, and so erected, cannot be otherwise than damp. The absence of any provision to prevent the moisture of the ground rising into the walls, and the thinness of the latter affording so little defence against rain, the interior can seldom or never be as dry as a house should be. From the want of subjacent ventilation any emanation from the soil must find immediate vent into the houses." The population is about 5,600 persons.

The first thing that strikes one in looking through the mortality tables is the large number of deaths from tubercular disease. 150 in five years in a population of 5,600—thirty each year. 5·3 per 1,000.

2. Although these deaths are scattered about throughout the district, about 15 per cent. take place in the narrow courts opening by passages into the streets.

3. The longest and widest streets are Jersey Street, with ten deaths, and George Leigh Street, with eight; but the number of deaths in these streets is approached by the mortality of eight in Hood Street, half their length—a short lane blocked at each end, Silk Street and Primrose Street, each with nine deaths. Henry Street, a long thoroughfare, has only four deaths, whilst Bond Street, a *cul-de-sac* one quarter its length, has seven.

4. The coincidences of deaths from phthisis in the same house within the space of five years are also most common in the more confined areas.

Thus there are two deaths in the same house at No. 2, Chapel Court, three at No. 13, Bond Street, and two at No.

18. There are three deaths at No. 10, Jepson's Court, and two at No. 3. Two at No. 3, Hewitt's Court; and in Hood Street there are two deaths each at three houses, Nos. 2,

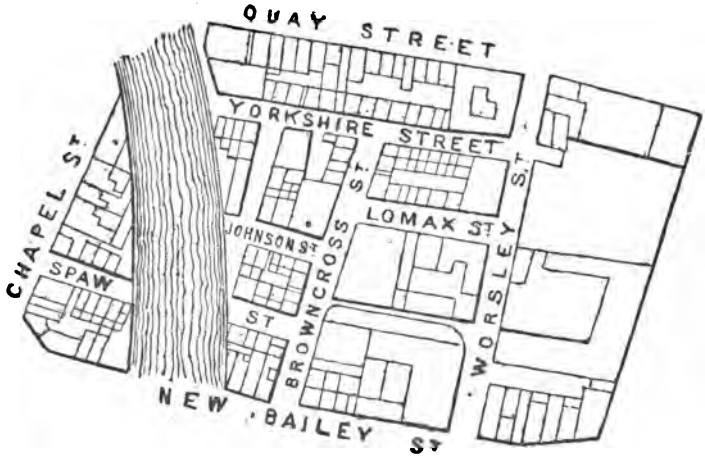
36, and 45. In Spittal Street there are two such coincidences, one at No. 12, and another at No. 29. In Gun Street there are two deaths at No. 43; in Blossom Street two at No. 52; and in Berwick's Court there are three at No. 5.

In Lorne Street there are two at No. 2. In the rest of the district there are, in Henry Street four cases of such coincidences, at Nos. 9, 20, 30, and 52; in Bengal Street one at No. 31; and in Jersey Street one at No. 29.

Altogether there are twenty-one such coincidences, or forty-four deaths, and fifteen of them (thirty in all) occur in the narrow streets, *culs-de-sac*, or small courts.

In Salford the districts into which it is divided are much smaller than those in Manchester, in fact they are the enumeration districts adopted at the last census; but Dr. Tatham,

ENUMERATION DISTRICT Nº 3.



the Medical Officer of Health, kindly selected three contiguous portions, with a total population of 2,609, and he gave me the particulars of their mortality, taken for the six years 1879-85, and had the accompanying map drawn for me (see p. 132).

Dr. Tatham also provided me with similar materials for another district, No. 3, Regent Road; and he personally visited the Greengate districts with me, and verified the causes of death recorded. I am greatly indebted to him for the kind interest that he took in my inquiry, and for the great assistance that he afforded to me.

It will be seen from the map that these districts are simpler in their construction than the Ancoats district. There are fewer courts and alleys, but the streets are almost wholly

composed of back-to-back houses; and whilst some of them are thoroughfares, others, notably Durham Street and Rylands Street, and Springfield Terrace or Place, are closed at one end. This construction enables us to compare the streets more readily, and we shall find that their respective phthisis-rates bear out the conclusions at which we have arrived with respect to Ancoats.

In Greengate district, No. 117, with 892 inhabitants, there are in the six years forty-seven deaths from tubercular disease, and of these twenty took place in Durham Street, which is blocked at one end, and nine in Rylands Street (on one side only), all in the single or back-to-back houses, and thirteen of these are in groups of two or more in the same house. In Broster Street, also entirely composed of back-to-back houses, and with its ventilation checked by a curve at the end nearest St. Simon Street, there are thirteen tubercular deaths, two of them at the same house, No. 66.

On the other side of Rylands Street, in No. 118 district, there were only two such deaths, and it is interesting to note, that although the cottages are just as small as the others, they have an outlet to the back.

In Bedford Street also, a broader street, mostly composed of similar cottages, opening at the back, there were only twelve tubercular deaths, and six of these were in the three houses Nos. 4, 11, and 41, in the worst part of the street; and in the whole of this No. 118 district, with 880 inhabitants, there were only 29 such deaths.

In No. 119 district, with 837 inhabitants, there are in all twenty-one tubercular deaths, and seven of these are in the short, narrow *cul-de-sac* called Springfield Terrace, two of them in one house, No. 9, whilst there is only one in the broad Robert Street, and one in Philip Street, and none in York Street,

These facts show that here, as in Ancoats, the streets most infected with phthisis are also the most confined and ill-ventilated, and that the larger proportion of deaths take place in the cave-like back-to-back dwellings.

Dr. Tatham writes, with regard to the subsoil:—"I have gathered from the surveyor, who has for years past been engaged in repairing the drains in this locality, that the subsoil underlying these districts is mainly clayey and alluvial. There is no accurate geological chart of the district."

In the Regent Road District, No. 3, numbering 791 inhabitants, there were in the six years forty deaths from undoubted tubercular disease, which is as high an average as that of Greengate, but the arrangement of the houses is less

regular, and there are many works interspersed amongst them. It is thus more difficult to make a comparison between the different parts of the district. There are also fewer back-to-back houses. Still, ten out of the forty cases occurred in five houses, *i.e.*, there were five coincidences of the disease in the same house, two of them in Quay Street, one in Shaw Street, one in Yorkshire Street, and one in a small court, Bennett's Square.

I am aware that there are certain sources of fallacy in drawing conclusions from these statistics. There is the possibility of wrong diagnosis, and, still more likely, there is the uncertainty as to whether the disease originated in the house in which the several deaths took place. In a working-class population, such as that in these districts, there are frequent changes of residence, and hence we cannot be sure that the same family has occupied the same house for any length of time.

I would point out, however, that errors in diagnosis may have been made in more than one direction, and that it is even more likely that a case of phthisis has passed undiscovered under the guise of bronchitis or pneumonia, than that the opposite mistake has been made.

The change of residence would also tell just as much against the view that I have taken of the results as in its favour, and, therefore, we may perhaps be allowed to set off one against the other.

There is yet another possibility, namely, that these poor sufferers might have drifted in their poverty from healthier houses into the cheap and unhealthy quarters in which they died. As a matter of fact, however, these work-people seldom leave the same district, though they may change their houses, and, as may be seen from the maps, the characters of the houses themselves is very much the same as regards accommodation, and their weekly rental varies very little in different streets.

On the whole I am inclined to accept the mortality tables as giving, if not an exact, yet a very fair, picture of the incidence of tubercular disease in the several parts of each district; and I venture to think, that if the areas which we have found so much infested with tubercle were opened out and improved, we should find a great diminution in the amount of this disease; and I claim them as, on the whole, supporting my hypothesis of the existence of specially-infected areas, in which either polluted ground air or an atmosphere reeking with organic matter have given virulence to the organised germ of the disease, much in the same

way as sewage, or polluted water, contribute to the conveyance of cholera and typhoid fever. And there is nothing in the natural history of tubercle that need run counter to this theory. Tubercle has been compared by Villemin to the granulations of glanders, and to the gummata of syphilis, and other observers have found analogies to it in the ulceration of Peyer's glands in enteric fever. It has further been shown, by Thiersch and Prof. Burdon Sanderson, that the virulence of the poison of cholera and typhoid fever increases after its extrusion from the body. There is, therefore, nothing unlikely in the hypothesis that contact with a certain kind of organic matter will assist the sporulation of the bacillus of tubercle, and render it more infective after a time than at the moment of its departure from the lungs of a phthisical patient.

It is on these grounds that I venture to propose my explanation of the mode in which tubercular diseases cling especially to certain localities, and to account for its spreading infectively in certain areas, whilst it is so rarely directly infectious from person to person.

DIPHTHERIA.

By GEORGE TURNER, M.D.

(*Read: June 8th*, 1887.)

GENTLEMEN,—I have occasionally heard a man, about to read a paper upon some subject, commence by expressing his sense of his unfitness for the task he has undertaken. I have, I must confess, looked upon this appeal, *ad misericordiam*, with much suspicion, believing the writer, did he not in his own heart believe himself particularly well-fitted for the work, would not have undertaken it. To-day, the fact that such a feeling may be very real is unpleasantly borne in upon me.

I have to speak of diphtheria, and find myself, in the vast majority of instances, unable to recognise the disease in the living subject. I mean, that out of a hundred cases in an epidemic I should be unable to diagnose any one out of perhaps eighty of them, were the patients placed before me singly.

Those whose experience is confined to hospital wards, or to the investigation of epidemic diphtheria, I believe hardly appreciate the difficulties which surround the diagnosis of that disease as it is seen by the general practitioner. I do not flatter myself that I can hold forth to you on the clinical part of my subject, and I am not labouring under the mistaken notion that this is the Clinical Society. But since the recognition of the disease is necessarily the first stage in any enquiry as to causes, it may be permitted me to ask if there can be anything more confusing than the description given us of the appearance noticed in the fauces: the colour is stated (not in the same work, but in half-a-dozen I have had the curiosity to consult) to vary from violet, through bright red, to "somewhat red". And they almost unanimously declare that the disease is characterised by the exudation of false membrane. If "characterised" is to mean, that membrane is met with in the majority of cases, with all due deference, I must dissent from such a definition; the greater number of cases never at any period develop a membrane. If by "characterised" is meant, that the presence of a mem-

brane is diagnostic, we are immediately plunged into the debated question as to the identity, or otherwise, of croup and diphtheria.

If, as I believe, the two disorders are essentially distinct, then the membrane ceases to be characteristic, and the diagnostic signs of diphtheria are still to be sought. The man who has the best opportunity of discovering them will be the general practitioner, because he sees a greater number of slight cases.

As I have frequently, and shall frequently, differ in opinion as to the nature of cases certified to have died of croup, it is only right that I should confess I am unable to distinguish isolated cases of tracheal diphtheria from croup.

Having made a clean breast of my ignorance, it is fair to inquire, how I can presume to contradict the opinion expressed by the medical man who has attended the patient all through, and has perhaps certified as to the cause of death? I do so, and arrive at the conclusion, that many cases of follicular tonsilitis and of croup are, in fact, diphtheria, on evidence I have been able to obtain, in certain instances, as to events preceding or following the attack, and not confined to the patient.

If some amongst us believe that diphtheria is frequently masked, we all of us agree that when it does declare itself, there can be no mistaking its identity. And it is because these slight cases of sore-throat so frequently give rise, in others, to diphtheria, concerning which there can be no two opinions, and *vice versâ*, and because so many cases certified as croup are preceded or followed by tonsilitis, that on the one hand I venture to call diphtheria many cases considered by the medical attendant to be tonsilitis, and on the other hand, despite my belief in the reality of croup as a distinct disease, I venture to differ in so many instances from the death certificates.

For example :—Diphtheria has of late years been prevalent in Aldershot. In 1883, thirty-two deaths were registered from throat sickness of all kinds. Of these, only nine were designated diphtheria. Of the twenty-one deaths from croup, no information could be obtained concerning five, because, as might be expected in the ever-shifting population of Aldershot, the families could no longer be traced. Twice, two fatal cases of croup occurred in the same house. In one, as far as I could ascertain, no other case of throat sickness had occurred in the family, but in all the others sore throats of varying intensity had preceded, or followed, the so-called croup.

The fact that two children died in the same house from croup, might be a suspicious circumstance, but could not be put forward as proofs that the disease had been infectious, a character it would not have possessed had it been croup. That sore throats had occurred contemporaneously with acute membranous trachitis would not be remarkable, but that only one instance should have been found in which fatal croup was not associated with some throat affection in the other members of the family, is very significant. Especially if taken in conjunction with the fact that nine deaths were registered as caused by diphtheria, in the same place, and during the same period; and that in the following year, diphtheria was prevalent and universally recognised.

In 1884, seventy-seven cases are known to have occurred in Aldershot, and forty-two deaths resulted. This year, diphtheria is usually given as the cause of death; thirty-one were returned under that head; and in 1885, when one hundred and twenty-six cases and forty deaths were discovered, the number ascribed to diphtheria is still greater, viz., thirty-six.

Are we to suppose that the medical practitioners at Aldershot were gradually being educated to recognise diphtheria? But then, in 1886—up to July—of thirteen deaths, three were registered as croup. Besides, those of them with whom I conversed upon the subject stoutly maintained that the early deaths were croup, the latter, diphtheria—that the disease was completely different at the beginning and at the end of the epidemic.

Although I differ from them as to the nature of the earlier cases, there is evidence that the type of disease had completely changed. For instance, in 1883, death, when it supervened, was brought about either because the membrane was confined to, or had extended into, the trachea, causing suffocation. Symptoms of blood-poisoning were not prominent, and paralytic sequelæ were confined to two exceedingly doubtful instances. In 1884, when the disease was more generally recognised as diphtheria, death by extension of the membrane into the trachea was still the more common fatal termination, but nine patients who recovered suffered from paralytic sequelæ, and death by cardiac failure appears to have happened in three instances. In 1885, cardiac failure was the immediate cause of five deaths, and twenty patients exhibited paralysis of the muscles of the throat or eye, or both.

In other words, during the earlier period, unless the patient was suffocated, he recovered; whereas later, the

symptoms which ushered in death pointed to some general poisoning of the system. The deaths in the first instance corresponded to the classical description of croup, and were registered as such.

The same change in the character of the disease may be noted to have occurred in other epidemics elsewhere. Hence, when called to investigate isolated cases attributed to croup, if I find that the other members of the family have been affected with sore-throat, I have usually no hesitation in stating my belief that the so-called croup was in fact tracheal diphtheria.

The idea that defective drainage and diphtheria are associated as cause and effect, is, perhaps, one of those most widely accepted by the medical profession. It has been too often our habit, when called to a case of diphtheria, to fix upon some defect in the sink or the water-closet, however trivial, and, having ascertained its existence, to rest more or less satisfied with the discovery. But diphtheria in the country occurs where such defects, from the position and surroundings of the dwellings are impossible, too often, to allow us to be satisfied with such an explanation. That bad drainage, however, does exert a considerable effect, influencing especially the course and result of the disease, is, I think, certain, and was exemplified at Aldershot. In that place, the Local Board had, just before my visit, caused an examination of the houses to be made with reference more especially to water-supply, drainage, etc. By aid of the tables thus provided, I was able to ascertain the proportion of drained and undrained houses which had been invaded.

Of the undrained houses, 24.6 per cent. were invaded. Of the drained, only 5.9 per cent. And, furthermore, it is to be noted of the drained houses in which diphtheria had occurred, I found, upon inquiry, that the drains were defective in 42 per cent. at the time the children were ill.

These proportions are very high, especially when it is remembered that the majority of houses were drained, and that diphtheria, unlike typhoid, is communicable from person to person directly. To save time, I will bring forward another cause, the probability of which has attracted much attention, viz., dampness,—and then consider it in conjunction with defective drainage.

At Portsmouth, in 1880, when the epidemic there was commencing, all the first cases, some twenty-five or so, resided in buildings against which no defects of drainage could be alleged, inasmuch as the water-closets were outside the houses, and there were no sinks. In fact, the

drains had no connection with these dwellings. They had only one peculiarity common to the whole of them—they were damp. It is a defect very frequently noticed in houses invaded by the disease, as is proved by the frequency with which it is mentioned in reports. And one would be tempted to suppose it was an essential condition, were it not that we meet with diphtheria epidemic in its worst form on high ground—gravelly soil with the ground water 50 ft. below the surface; and in the neighbouring valley, perhaps, where the subsoil water is at a spade's depth, the disease is absent or nearly so.

Probably dampness of the dwelling assists the disease by conducing to simple sore-throat; and diphtheria, in one respect, resembles erysipelas,—it enters the system more easily through an abraded surface than through the healthy mucous membrane. Just as the dresser whose cuticle is intact goes about his work in the erysipelas ward and escapes, but would almost assuredly take the disease were he to apply a small blister to some part of his skin, so the person whose throat is healthy appears to offer less opportunity to the virus than one who is suffering from an inflamed or congested state of his throat. Hence the history of diphtheria cases often commences with the record of a cold, or sore-throat, caught by getting wet through, etc.

This predilection for an abraded surface may account for the fact that, in not a few instances, the first symptoms of which the child has complained, indicated that the Eustachian tube, or the internal ears, have been affected, the child suffering at the time probably from catarrh of that organ. It is noticeable, too, that diphtheritic membrane round the teeth is less frequently observed now than it appears to have been in the days of Betonneau. I have only seen it in one instance, and its present rarity may be connected with the better state of the gums, now that teeth are more regularly cleaned and attended to. The infrequency with which the membrane attacks the wound in tracheotomy, seems to indicate that it prefers a mucous membrane to any other raw surface.

To return to the condition of the houses: whenever dampness is produced by the impregnation of the soil with water containing sewage in solution, its pernicious effects seem to be much more certain. This state of things was observed at Hale, where the subsoil water was 50 ft. below the surface, and at Ash also. The soil was very porous, and the privies were, in the majority of instances, so situated, that the contents of the privy-pit must have percolated under the buildings.

At Ash, this was noticeable in the houses inhabited by families in which the worst cases occurred. In one instance, out of ten persons, members of the household, seven were attacked, and five died. Probably the others would have suffered had they not been moved to the wards for infectious disease at the union. The house was situated on the side of a high sand hill, the water in the well was 30 ft. below the level of the cottage floor, yet the place was damp, not only from rain-water running down the surface of the hill towards the building, but because the privy was against the house, so that the contents of the cess-pit percolated into the sand upon which the dwelling was built. The same kind of arrangement was met with at Hale, at Farnborough, and Farnham. At the latter town, a very good contrast was afforded by the houses on the two sides of the main street. On the one side were houses with common privies closely adjoining them, in one instance actually inside the dwelling. There fatal sore-throat sickness had been common. On the other side, where the privies were away from the dwellings, and where, from the relative positions of the two buildings and the slope of the ground, or from the use of earth closets, the soil could not be impregnated with sewage, there fatal diphtheria was absent, and sore-throat rare. The same thing was observed at Aldershot and Petersfield.

Given the introduction of a case of infectious sore-throat into a neighbourhood in which such conditions are met with, it is easily understood that the disease would spread. Of course, in many cases we cannot expect to find all, or any, of the surroundings indicated. The fact that diphtheria is communicable from person to person would be certain to give rise to the disease, amongst people living in houses which from a Health Officer's point of view are perfect; just as we should find small-pox or scarlet fever in them.

Undoubtedly, the schools are responsible, in a very great measure, for spreading the disease, and account for the recurrences we so frequently observe. The fact that many people reside in one small cottage is against the chance of any of them escaping infection, and, having become infected, it is conducive to a fatal result. At Aldershot, the population was 8.3 per house, and, speaking generally, those villages which showed a high average population per house, *ceteris paribus*, show a high mortality from diphtheria when it is epidemic in them. This was seen in the camp at Aldershot, when the accommodation for the families of soldiers, although in other respects good, was limited as to cubic space.

When many children are brought together in one room, as

at a board school, the means of infection are multiplied, and in country places, where some of the children must walk long distances in all weathers, and sit in wet boots and damp clothes, every facility is offered for the reception of the poison. It is, I suppose, an open question whether the assemblage of several cases in the same room intensifies the poison or affords more frequent opportunities for infection. Certainly, it is a fact that slight cases of sore-throat may prevail in the cottages without giving rise to very serious results, but no sooner do the children meet in school, after it is re-opened, than diphtheria makes its appearance amongst them.

This was very evident at Frimley near Aldershot. And lately at Brent Pelham, in my district, diphtheria broke out in one cottage; four children were attacked; they were removed from the village; one died, and the others recovered. After fifty days, they returned to school, and within forty-eight to seventy-two hours other cases appeared amongst the scholars. There is no doubt these children came back to school at too early a period of their convalescence. The same misfortune resulted from similar causes at Little Hallingbury. It is a matter of importance that we should have a clearer notion as to the period at which children who have been suffering from sore-throat can be allowed to return to school. That they are dismissed from hospital much too soon is evident, because, if the number of instances in which paralytic sequelæ are observed, according to the reports of hospital cases, be compared with the frequency with which they are noted by the Medical Officer of Health, it will be found they are far more common than is usually supposed. In the greater number of instances, a period of comparatively good health intervenes between the acute attack and the diphtheritic paralysis, and in that interval the children are discharged. Probably, while in that condition they are infectious.

I believe I have seen the disease spread by drinking water on more than one occasion; twice it has been conveyed by a corpse; once it was communicated by some clothes which had been shut up for three months in a drawer; and it has been clearly proved to have been spread by milk.

Admitting all these means of dissemination, we meet with numerous cases in which the closest investigation fails to bring to light any of them, and the problem as to how the patients were infected remains unsolved. I would suggest, that one way in which infection is conveyed is through the lower animals. The possibility of this has occupied my attention since 1882; and, although I have been unable to

prove the truth of the supposition, a certain amount of evidence has been obtained, which, it is hoped, makes the question worthy of consideration.

The conveyance of disease through milk is now commonly recognised; but, until lately, the disease has been transmitted from man to man by the milk, and not from the cow to the man. Mr. Power, Dr. Cameron, and Dr. Klein, have recently shown that in the case of scarlet fever the disease may originate in the cow, and be communicated to those who drink her milk; and, moreover, that the bovine malady may be a comparatively mild disorder, and escape the notice of those who attend the animals.

With infectious diseases, we cannot argue too closely from one variety to another; those which are apparently most closely allied differ greatly as to incubation period, power of infection, period at which the disease is most infectious, and duration of infective power,—measles and scarlet fever for example. Beyond this, since the days of Jenner, the variation in the behaviour of the same virus in different animals has been daily before us, yet its significance seems to have been more clearly understood by Jenner himself than by the majority of those who succeeded him.

Small-pox, highly infectious and fatal to man, is no longer very fatal, or so infectious, when communicated to the cow, for the milkers seem to be infected by actual contact only, and the attack is never fatal. The disease, when returned to the soil in which, in its unmodified state, it was both infectious and fatal, does not, after even a long series of cultivations, recover its lost qualities, and, but for the protection it affords, there is little to indicate that it is more than a local affection—the behaviour of this well-known virus demonstrates that.

The inoculation of an animal with a material suspected of being the cause of a specific disorder in another species, when it produces no effect, or when it is followed by a disease no longer resembling the first, and when this induced disease, being returned by inoculation to the class of animal from which the original material was obtained, fails to produce any effect, or produces a disease no longer resembling the first; all, or any of these occurrences may be compatible with the suspected material being the virus of the disease first in question. It may mean, that the virus has been modified in transmission; and the possibility of modification must always be present in the minds of those who investigate the causes of infectious disease.

In 1882, a pigeon was brought to me; it was supposed to

have died of strangles (*sclerostoma cyngamus.*) I dissected it, hoping to obtain some specimens of that worm. Instead, I found the trachea lined with a consistent membrane, looking like those seen in the bodies of children dead of croup.

Pigeons were inoculated with this membrane by a man who takes some interest in such experiments, and they suffered in a similar way, excepting that the membrane was not in any case so well-developed, and in most consisted of masses of epithelium caked together. The disease passed up the nostrils, and invaded the conjunctiva.

A medical man inoculated a pigeon with some membrane obtained from the throat of a child, and no result followed; he would have supposed that the disease was not communicable from the human being to the bird, had he not, at the same time, inoculated some cultivation tubes, which remained sterile. He made inquiries, and found that the membrane had been removed from the throat with a brush dipped in sulphurous acid.

A piece of membrane was procured from another patient, which had not been interfered with—this produced the disease in pigeons.

In 1883, an epidemic of diphtheria in the village of Braughing was carefully investigated. The first case was that of a lad who worked at a farm where the chicken were dying of diphtheria, and the earlier cases were all in the families of persons employed upon the farm, or who obtained milk there. The disease made its appearance in another farm, where it spread in a similar manner.

I have had occasion to notice the connection between fowl-diphtheria and human-diphtheria in other instances,—at Manuden, at Aldershot, at Farnham, at Ash, at Tungham, and at Long Eaton. Similar accounts are received from abroad, so that the identity and transmissibility of the disease in man and fowls seems probable.

I had noticed diphtheria amongst swine on one occasion, but the disease did not then spread amongst human beings, and my attention was directed principally to birds. I found it amongst pigeons, chicken, pheasants, and turkeys, but have as yet never observed it, or heard of it, amongst ducks or geese. Care must be taken not to confound the account one receives of deaths amongst these creatures which are due to strangles. Usually the young chicken are affected by the worm, but the older ones die from diphtheria.

During January 1886 I had to investigate diphtheria at Brent Pelham. The disease was confined to one family. The village is a small one, and I made a careful search for throat-

sickness amongst the children, without any result. The cat, however, at this cottage had died, with running from the eyes and nose, and swelling at the angles of the jaws. In all, nineteen of these animals had met with a similar fate. I offered 10*s.* 6*d.* for one of them, but the owner preferred knocking it on the head to letting me have it.

The cats had died during the epidemic of diphtheria at Farnham, at Aldershot, Yateley, and Petersfield. At the latter place the evidence was particularly strong, not that the children took the disease from the cat, but that the animal was infected by the child.

At Moulton, some children sick of diphtheria were confined to the upper part of a cottage, away from the rest of the village, by order of the medical attendant, that the rest of the family might have a better chance of escaping. The attempt at isolation succeeded; but the cat, which was fed with food left by the sick children, suffered.

In the *British Medical Journal*, Jan. 3rd, 1885, there is an account of some experiments made by C. J. Renshaw, M.D., who appears to have succeeded in inoculating cats with diphtheria from the human subject.

It is a well-known fact that horses suffer from sore throats, certain varieties of which are called strangles, but veterinary surgeons have not as yet recognised diphtheria amongst horses.

I do not suppose that all strangles are diphtheria, any more than all sore throats amongst us are diphtheria, but I think it possible, one of its forms may be connected with human disease.

At Moulton, the first case of diphtheria occurred in the family of a farmer, shortly after a horse had died of the strangles. The second case was the man who had charge of the horse. In the neighbouring village of Ousden, a man who had recently recovered from diphtheria was employed to groom a mare; the animal within a few days suffered from sore-throat, swelling at the angle of the jaws, and a foul discharge from the nostrils. In Yateley, in two instances, the human disease was coincident with strangles amongst the horses. At Hormead, the only case occurred in the house of a farmer whose horses had recently died of strangles.

If the nature of the employment pursued by those who die of the disease, or in the case of children that of their parents, be noted, it is surprising what a large proportion are persons following occupations connected more or less with horses, or other of the lower animals.

Dr. Ogle informs me he met with one instance in which

diphtheria appeared to be connected with disease amongst the sheep, and a similar case is related in one of the reports of the Medical Officers of the Local Government Board. I was asked to see a flock of sheep which were affected with a disease of the throat in a village in which diphtheria was prevalent, but the disorder was, in my opinion, foot-and-mouth disease. The shepherd, however, did not agree with my view of the case.

I think, on the whole, there is ample evidence to justify a careful inquiry into the connection which may exist between human diphtheria and sore-throat in the lower animals. It would, if confirmed, go far to explain the occurrence of diphtheria in places where, as in the Australian bush, conveyance by human beings seems out of the question, and might throw some light upon the variations observed in the mortality met with in different epidemics.

DR. NORMAN CHEVERS, C.I.E.

DR. NORMAN CHEVERS, whose death the Society has to deplore, was for many years a distinguished member of the Indian Medical Service. The son of a naval surgeon, F. M. Chevers, who had been in action in Lord Howe's famous victory, and under Nelson at Trafalgar, he was early initiated into the medical profession, first in the wards of the Royal Naval Hospital, Haslar, and subsequently in those of Guy's Hospital. He then proceeded to Glasgow, where he graduated in 1839, at the age of twenty-one. Returning to London, he was for some years actively engaged in pathological research, chiefly into the structure of the heart and blood-vessels, the results of which were published in the Guy's Hospital Reports, and in the *London Medical Gazette*. He was also one of the original members of the Pathological Society. Of his numerous excellent papers on morbid anatomy at this time, the most noted perhaps was that "On the Causes of Death after Operations and Injuries in London Hospitals," deduced from the history of 153 fatal cases at Guy's. It was quoted in terms of high commendation by Sir James Simpson in more than one of his works.

Having thus acquired a series of valuable observations and a large experience in the Metropolis, it is not surprising that when, in 1848, Dr. Chevers, at a maturer age than most medical officers, accepted an appointment on the Bengal establishment, he at once took a high position in the scientific medicine of India. He was Secretary for some years to the Medical Department, for two years he officiated as the Inspector-General of Gaols in Bengal, and for fifteen years he was Principal of the Calcutta Medical College, and Professor of Medicine and Senior Physician of the College Hospital. During his tenure of those offices, the number of medical students increased from 409 to 1441, a signal proof of his administrative ability and attractive power as a teacher. Dr. Chevers' writings were very numerous, and on many topics, but his high reputation was greatly augmented by various publications on hygienic and other subjects of general interest, which acquired a large circulation, and were soon regarded as standard works of the highest authority.

Such were his *Preservation of the Health of Soldiers in India; Removable and Mitigable Causes of Death;* and *Medical Jurisprudence.* For the last work, Dr. Chevers was awarded, in 1879, the Swiney Prize, a silver cup of the value of £100, and also the sum of £100, given by the Society of Arts and the London College of Physicians, for the best work on Medical Jurisprudence published during the preceding ten years.

In 1876, under the altered regulations as to age consequent on the transfer of the Empire to the Crown, he retired from the Indian Service with the rank of Deputy Surgeon-General, returned to England, and settled in London. Although not engaged in practice, Dr. Chevers continued to take the warmest interest in all matters pertaining to the medical sciences, especially in those which bear on public health. He was for some years an active member of the Epidemiological Society, and for two years (1883-5) filled the office of President. His extensive knowledge, wide and varied experience, and profound learning, were united to a happy lucidity of expression and a courteous urbanity of manner which greatly contributed to the success of the meetings. His distinction and special authority in Indian medicine attracted to the Society many eminent officers who had served in that Empire, and the discussions under his auspices were often of a very interesting and practical character by reason of the accumulated local experiences of the speakers. Dr. Chevers had been for several years much occupied on what has unhappily proved to be his last, but which is probably his greatest, work—*A Commentary on the Diseases of India,* which was published only a few months before his lamented death. He dedicated the volume to Mr. Alexander Grant, Honorary Surgeon to the Queen, his most intimate friend and fellow-labourer in his earlier years in Bengal, and joint-editor of the *Indian Annals of Medical Science,* "in remembrance of an unbroken friendship of thirty-five years, and in admiration of his masterly knowledge of Indian disease." The work is a monument of professional zeal, deep research, and indefatigable industry, which will maintain a lasting and increasing reputation as the outcome of the life-experience of an acute and thoughtful mind, and as a repertory of the medical science of the time, accumulated, to use again Dr. Chevers' words, by "the noble body of physicians and surgeons in association with whom it was my great privilege to spend the years of my active official career." The enormous labour involved in this undertaking, and in the arrangement of a huge mass of materials in the excellent literary form he knew so well to

give them, doubtless told injuriously on Dr. Chevers' health. Symptoms of increasing debility caused at times anxiety to his family, but his death was sudden and unexpected, from failure of the heart's action, on the 2nd of December 1886. He was interred at Kensal Green, and among the mourners were several of his Indian colleagues and members of the Society. A man of sterling worth, of great qualities, both of head and heart, Dr. Chevers' memory will long be held in affectionate regard by all who had the privilege to know him. In the words of the eulogy which appeared at the time in a professional journal, "His was indeed a blameless and noble life, one in which commanding intellect and vast stores of learning of many and varied aspects were devoted to the advancement of knowledge, the relief of suffering, and the welfare of his fellow men, especially of those of the great Indian Empire in which his lot had been cast, his best work done, and his well-merited reputation acquired."

The Ordinary Meetings of the Society are held at

11, CHANDOS STREET, CAVENDISH SQUARE,

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